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ARIZONA MEDICINE

JOURNAL OF ARIZONA MEDICAL ASSOCIATION

MEDICAL SOCIETY OF THE UNITED STATES AND MEXICO

Annual Meeting Highlights

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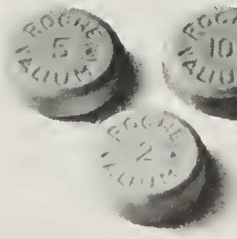
A character all its own.



Valium (diazepam/Roche) is a benzodiazepine with a character all its own. Pharmacologically, it is a potent skeletal muscle relaxant and anticonvulsant (in adjunctive use), as well as an antianxiety agent. Pharmacokinetically, only Valium provides active *diazepam* as well as the active metabolites 3-hydroxydiazepam, desmethyldiazepam and oxazepam.

But the individual character of Valium is even more apparent clinically than pharmacokinetically. And far more significant. That's because of the patient response obtained with Valium. A response which brings a calmer frame of mind. A response which has a pronounced effect on the somatic symptoms of anxiety, particularly muscular tension. A response which helps the patient feel more like himself again because of the way Valium reduces the overwhelming symptoms of anxiety and psychic tension.

Another important aspect of the clinical character of Valium is safety. Though drowsiness, ataxia and fatigue are possible, these and more serious side effects are rarely a problem. Of course, as with all CNS-acting drugs, patients taking Valium should be cautioned against driving, operating dangerous machinery or the simultaneous ingestion of alcohol.

Unquestionably, many psychotherapeutic agents, including other benzodiazepines, have antianxiety effects. But one fact remains: you get a certain kind of patient response with Valium. It's a response you want. A response you know. A response you trust as part of your overall management of anxiety and psychic tension.

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Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® (diazepam) Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10.

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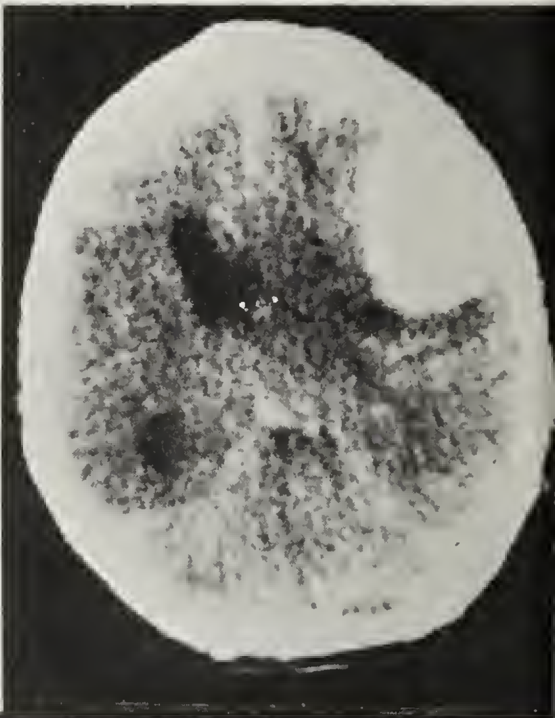

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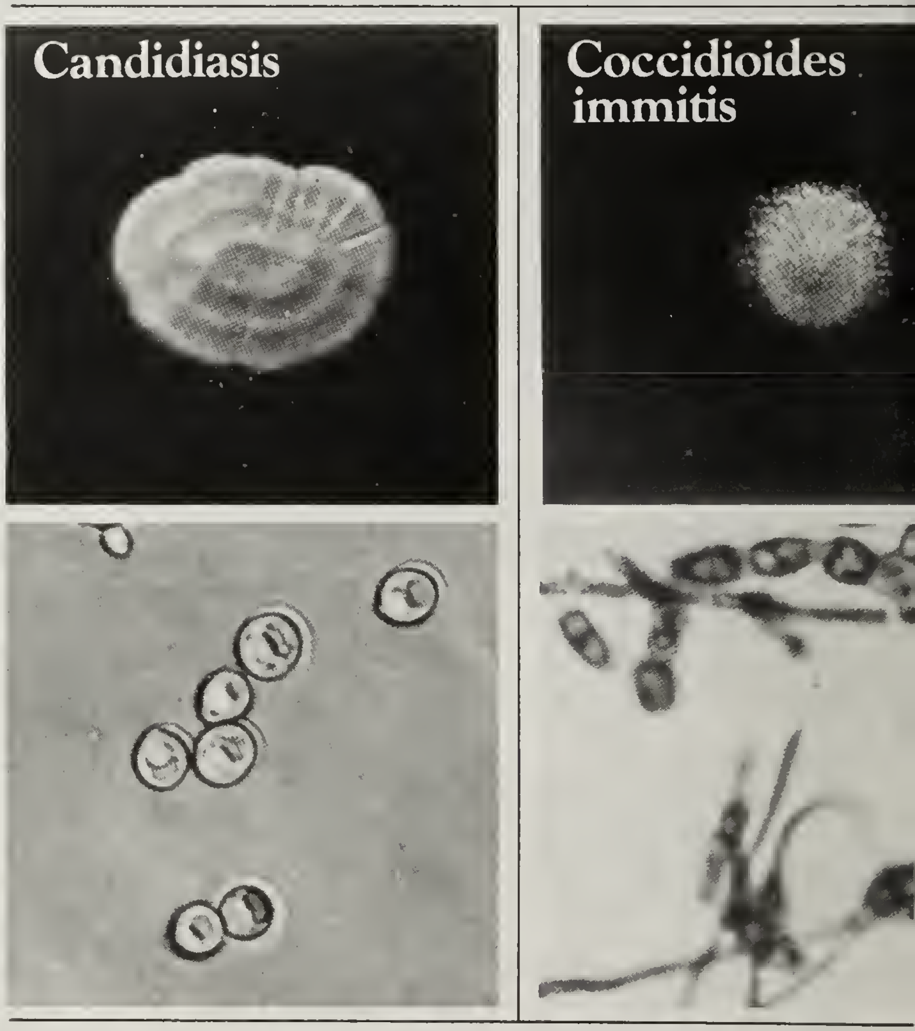
  - coccidioidomycosis
  - cryptococcosis
  - paracoccidioidomycosis
- in patients who did not respond to amphotericin B therapy
- in immunologically-compromised patients with severe infections

rapid onset of clinical improvement
In most patients who responded, improvement was observed within 1-3 weeks of MONISTAT i.v. therapy; however, prolonged treatment courses may be necessary to prevent relapse.

no development of fungal resistance during therapy reported
In vitro resistance of clinical isolates has not been observed during clinical trials.



Photos courtesy of Dubos, J. R., Bacterial and Mycotic Infections of Man, J. B. Lippincott Company, 1968 and Burrows, W., Textbook of Microbiology, W. B. Saunders Company, 19th editi

and the side effect profile

no nephrotoxicity or other permanent adverse effects in clinical studies

- even in prolonged therapy
- even in patients with renal disease

extensively evaluated
During clinical trials in over 400 patients, no permanent adverse reactions were reported.

Adverse reactions
MONISTAT i.v. should not be administered undiluted. Rapid injection of the undiluted solution may produce transient tachycardia or arrhythmia.

Adverse reactions include phlebitis, pruritus, rash, nausea, vomiting, febrile reactions, drowsiness, diarrhea, anorexia and flushes. Transient hematologic changes have occurred; therefore, the patient should be closely monitored.

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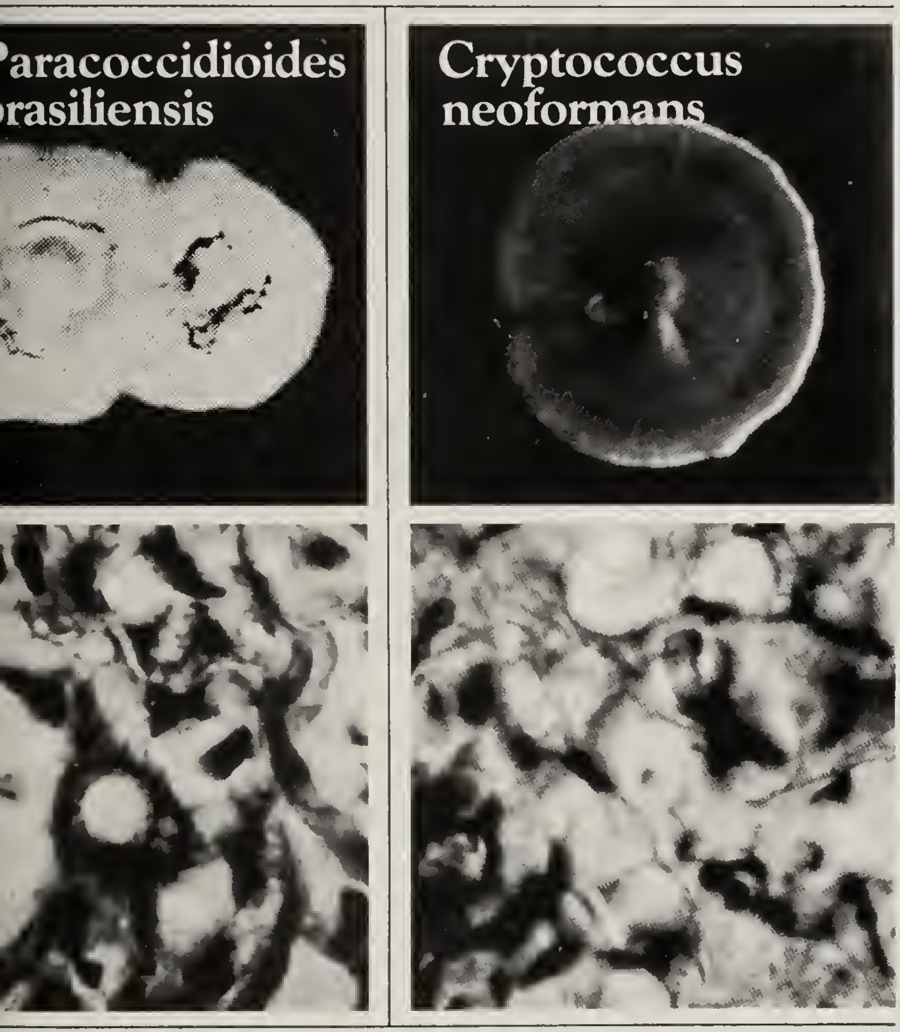
advancement
systemic mycoses



generally well tolerated by severely debilitated patients

- cancer patients
- patients receiving immunosuppressive drugs
- patients with serious underlying conditions such as renal insufficiency or diabetes mellitus

for more information

Because of the uniqueness of new MONISTAT i.v. only some of the highlights can be presented in this advertisement. For more information, i.e., product monograph, dosage schedules, etc., please fill out the coupon on the next page and mail to Ortho Pharmaceutical Corporation.

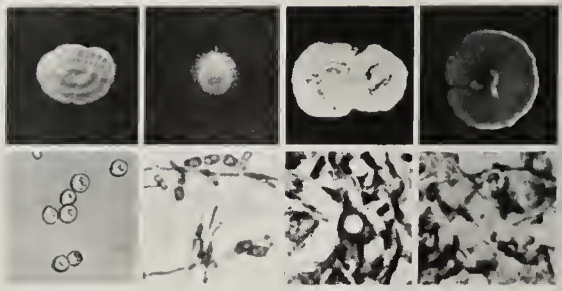
Please turn page for Prescribing Information

Ortho Pharmaceutical Corporation
Raritan, New Jersey 08869

new
Monistat i.v.
TRADEMARK
(miconazole)

broad-spectrum activity against major systemic fungal pathogens

in systemic fungal infections…



new Monistat i.v.

TRADEMARK

(miconazole)

a broad-spectrum systemic antifungal agent

DESCRIPTION: MONISTAT i.v. (miconazole) 1-{2-(2,4-dichlorophenyl)-2-[(2,4-dichlorophenyl) methoxyl ethyl} 1 H-imidazole is a synthetic antifungal supplied as a sterile solution for intravenous infusion. Each ml of this solution contains 10 mg of miconazole with 0.115 ml PEG 40 castor oil, 1.0 mg lactic acid USP, 0.5 mg methylparaben USP, 0.05 mg propylparaben USP in water for injection. Miconazole i.v. is a clear, colorless to slightly yellow solution having a pH of 3.7 to 5.7.

CLINICAL PHARMACOLOGY: MONISTAT i.v. is rapidly metabolized in the liver and about 14% to 22% of the administered dose is excreted in the urine, mainly as inactive metabolites. The pharmacokinetic profile fits a three-compartment open model with the following biologic half-life: 0 4, 2.1, and 24 1 hours for each phase respectively. The pharmacokinetic profile of MONISTAT i.v is unaltered in patients with renal insufficiency, including those patients on hemodialysis. The in/vitro antifungal activity of MONISTAT i.v. is very broad Clinical efficacy has been demonstrated in patients with the following species of fungi: *Coccidioides immitis*, *Candida albicans*, *Cryptococcus neoformans*, *and Paracoccidioides brasiliensis*.

Recommended doses of MONISTAT i.v. produce serum concentrations of drug which exceed the in-vitro MIC values for the fungal species noted above. Doses above 9 mg/kg of MONISTAT i.v. produce peak blood levels above 1μg/ml in most cases. The drug penetrates into joints.

INDICATIONS: MONISTAT i.v. is indicated for the treatment of the following severe systemic fungal infections coccidioidomycosis, candidiasis, cryptococcosis, paracoccidioidomycosis, and for the treatment of chronic mucocutaneous candidiasis. However, in the treatment of fungal meningitis and urinary bladder infections an intravenous infusion alone is inadequate. It must be supplemented with intrathecal administration and bladder irrigation. Appropriate diagnostic procedures should be followed and MICs should be determined

MONISTAT i.v. should not be used to treat common trivial forms of fungal diseases.

CONTRAINDICATIONS: MONISTAT i.v. is contraindicated in those patients who have shown hypersensitivity to it

WARNINGS: Rapid injection of undiluted MONISTAT i.v., may produce transient tachycardia or arrhythmia

PRECAUTIONS: Before a treatment course of MONISTAT i.v. is started, the physician should make sure that the patient is not hypersensitive to the drug product MONISTAT i.v. should be given by intravenous infusion. The treatment should be started under stringent conditions of hospitalization but subsequently may be given to suitable patients under ambulatory conditions with close clinical monitoring. It is recommended that an initial dose of 200 mg be given with the physician in attendance. It is also recommended that clinical laboratory monitoring including hemoglobin, hematocrit, electrolytes and lipids be performed.

It should be borne in mind that systemic fungal mycoses may be complications of chronic underlying conditions which in themselves may require appropriate measures.

Ortho Pharmaceutical Corporation **AM-7-79**
c/o 733 Summer Street
Stamford, CT 06901

Please send me more information on the new broad-spectrum, systemic antifungal agent…MONISTAT i.v. (miconazole).

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Specialty_____________________________

October 1978

Pregnancy: Reproductive studies with MONISTAT i.v. in rats and rabbits revealed no evidence of impaired fertility or harm to the fetus. There are no data, however, on the use of the drug in pregnant women.

Children: Since the safety of miconazole i.v. in children under one year of age has not been extensively studied, its benefits in this age group must be weighed against the possible risks involved.

ADVERSE REACTIONS: Adverse reactions which have been observed with MONISTAT i.v therapy include phlebitis, pruritus, rash, nausea, vomiting, febrile reactions, drowsiness, diarrhea, anorexia and flushes. In the U.S. studies, 29% of 209 patients studied had phlebitis, 21% pruritus, 18% nausea, 10% fever and chills, 9% rash, and 7% emesis. Transient decreases in hematocrit and serum sodium values have been observed following infusion of MONISTAT i.v. Thrombocytopenia has also been reported No serious renal or hepatic toxicity has been reported If pruritus and skin rashes are severe, discontinuation of treatment may be necessary. Nausea and vomiting can be mitigated with antihistaminic or antiemetic drugs given prior to MONISTAT i.v. infusion, or by reducing the dose, slowing the rate of infusion, or avoiding administration at mealtime

Aggregation of erythrocytes or rouleau formation on blood smears has been reported. Hyperlipemia has occurred in patients and is reported to be due to the vehicle. Cremophor EL (PEG 40 castor oil)

DRUG INTERACTIONS: Drugs containing cremophor-type vehicles are known to cause electrophoretic abnormalities of the lipoprotein. These effects are reversible upon discontinuation of treatment but are usually not an indication that treatment should be discontinued

Interaction with the coumarin drugs resulting in an enhancement of the anticoagulant effect has also been reported In cases of simultaneous treatment with MONISTAT i.v. and coumarin drugs, the anticoagulant effect should be carefully titrated since reductions of the anticoagulant doses may be indicated

DOSAGE AND ADMINISTRATION

DOSAGE

Adults. The doses may vary with the diagnosis and with the infective agent, from 200 to 1200 mg per infusion. The following daily doses, which may be divided over 3 infusions, are recommended:

Organism	Dosage Range*	Duration of Successful Therapy (weeks)
Coccidioidomycosis:	1800 to 3600 mg per day	3 to > 20
Cryptococcosis:	1200 to 2400 mg per day	3 to > 12
Candidiasis:	600 to 1800 mg per day	1 to > 20
Paracoccidioidomycosis:	200 to 1200 mg per day	2 to > 16

* May be divided over 3 infusions.

Repeated courses may be necessitated by relapse or reinfection.

Children A total daily dose of about 20 to 40 mg/kg is generally adequate. However, a dose of 15 mg/kg body weight per infusion should not be exceeded.

ADMINISTRATION. MONISTAT i.v. should be diluted in at least 200 ml of fluid. The diluent of choice is 0.9% sodium chloride or alternatively Dextrose 5% injectable solution. The intravenous infusion should be given over a period of 30 to 60 minutes.

Generally, treatment should be continued until all clinical and laboratory tests no longer indicate that active fungal infection is present. Inadequate periods of treatment may yield poor response and lead to early recurrence of clinical symptoms. The dosing intervals and sites and the duration of treatment vary from patient to patient and depend on the causative organism.

OTHER MODES OF ADMINISTRATION: Intrathecal. Administration of the undiluted injectable solution of MONISTAT i.v. by the various intrathecal routes (20 mg per dose) is indicated as an adjunct to intravenous treatment in fungal meningitis. Succeeding intrathecal injections may be alternated between lumbar, cervical, and cisternal punctures every 3 to 7 days. Bladder instillation of 200 mg of diluted solution is indicated in the treatment of mycoses of the urinary bladder.

HOW SUPPLIED: MONISTAT i.v. is supplied in 20 ml ampoules.

MONISTAT i.v. (miconazole) is an original product of Janssen Pharmaceutica N.V., Belgium, and co-developed by Ortho Pharmaceutical Corporation.

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Tenuate Dospan®
(diethylpropion hydrochloride NF) controlled-release

AVAILABLE ONLY ON PRESCRIPTION
Brief Summary
INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below.
CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states. Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).
WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly. *Drug Dependence.* Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia *Use in Pregnancy*: Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children*. Tenuate is not recommended for use in children under 12 years of age.
PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes, mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.
ADVERSE REACTIONS: *Cardiovascular*: Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System*: Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal*: Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic*: Urticaria, rash, ecchymosis, erythema. *Endocrine*: Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System*: Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous*: A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.
DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg tablet three times daily, one hour before meals, and in midevening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg. tablet daily, swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.
OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of April, 1976

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References: 1. Citations available on request — Medical Research Department, MERRELL RESEARCH CENTER, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215. 2. Hoekenga, M.T., O'Dillon, R.H., and Leyland, H.M.: A Comprehensive Review of Diethylpropion Hydrochloride. International Symposium on Central Mechanisms of Anorectic Drugs, Florence, Italy, Jan. 20-21, 1977.

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(diethylpropion hydrochloride NF)

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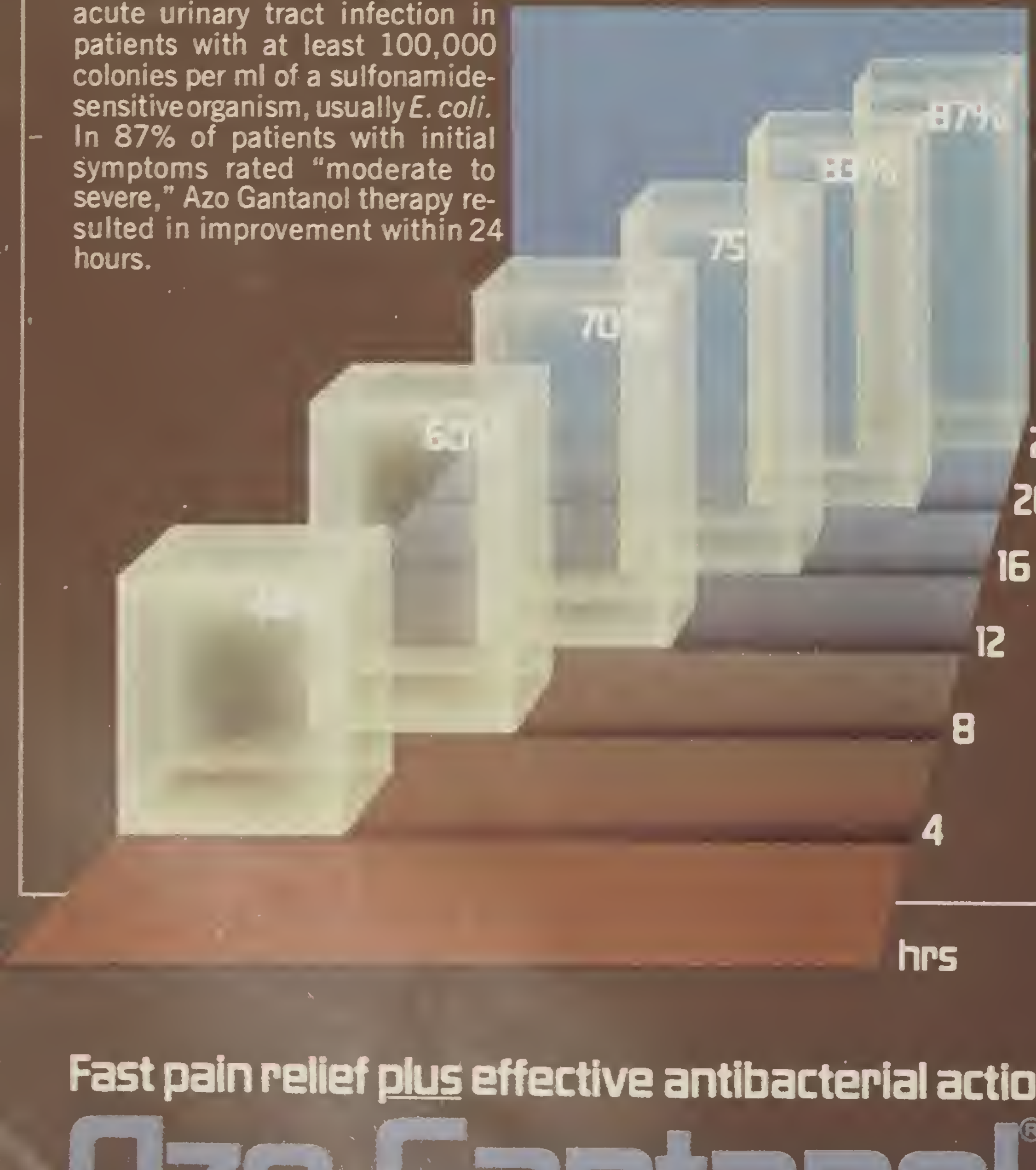

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uremia, and pyelonephritis of pregnancy w
disturbances.
Warnings: Safety during pregnancy not esta
Deaths from hypersensitivity reactions, a
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have been reported and early clinical signs
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stone formation.
Adverse Reactions: *Blood dyscrasias* (agran
ulocytosis, aplastic anemia, thrombocytope
leukopenia, hemolytic anemia, purpura, hy
thrombinemia and methemoglobinemia); *al*
reactions (erythema multiforme, skin erupt
Stevens-Johnson syndrome, epidermal ne
urticaria, serum sickness, pruritus, exfoliat
dermatitis, anaphylactoid reactions, perior
edema, conjunctival and scleral injection,
sensitization, arthralgia and allergic myoca
G.I. reactions (nausea, emesis, abdominal
hepatitis, diarrhea, anorexia, pancreatitis a
stomatitis); *CNS reactions* (headache, peri
neuritis, mental depression, convulsions, a
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miscellaneous reactions (drug fever, chills,
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nodosa and L. E. phenomenon). Due to ce
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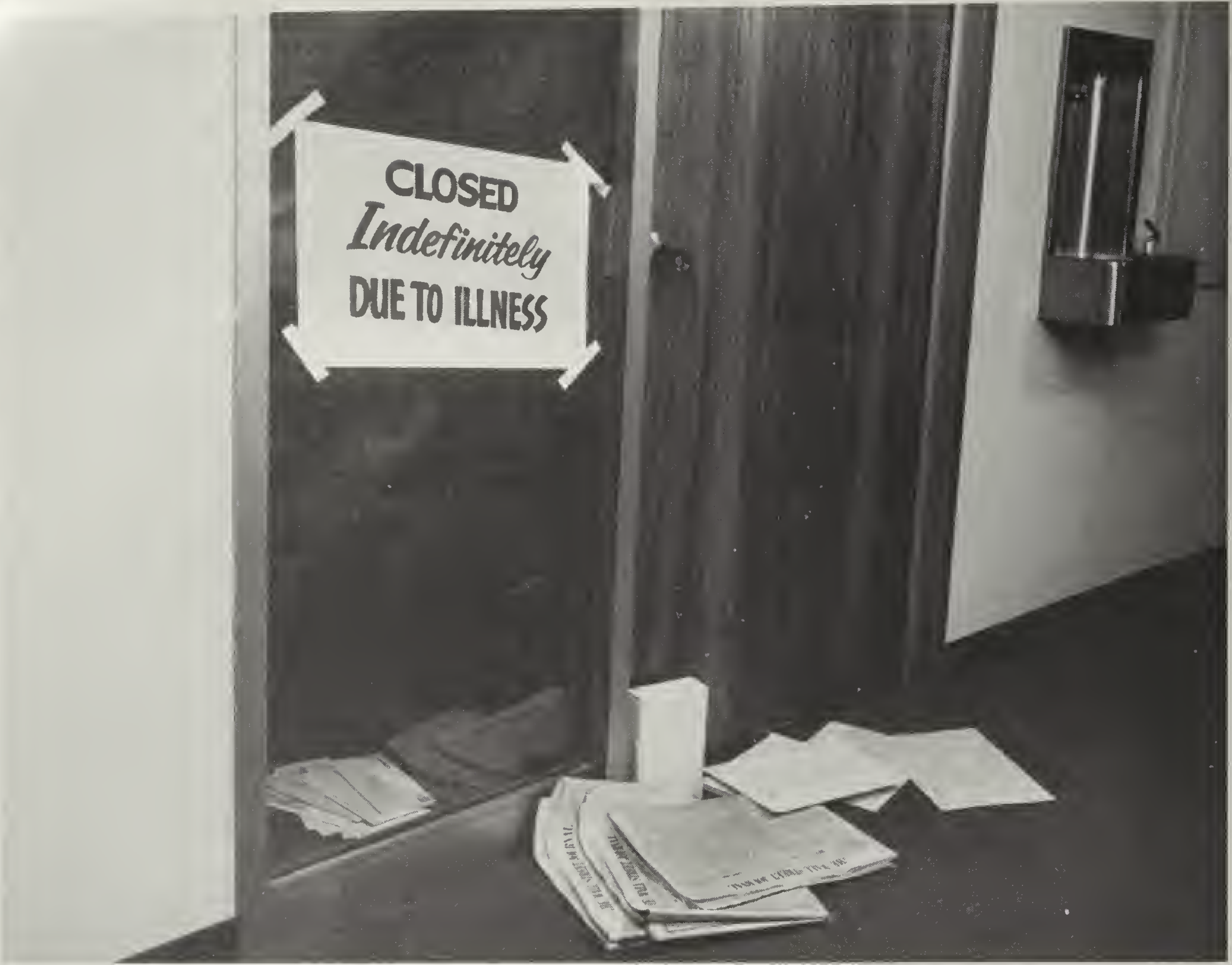

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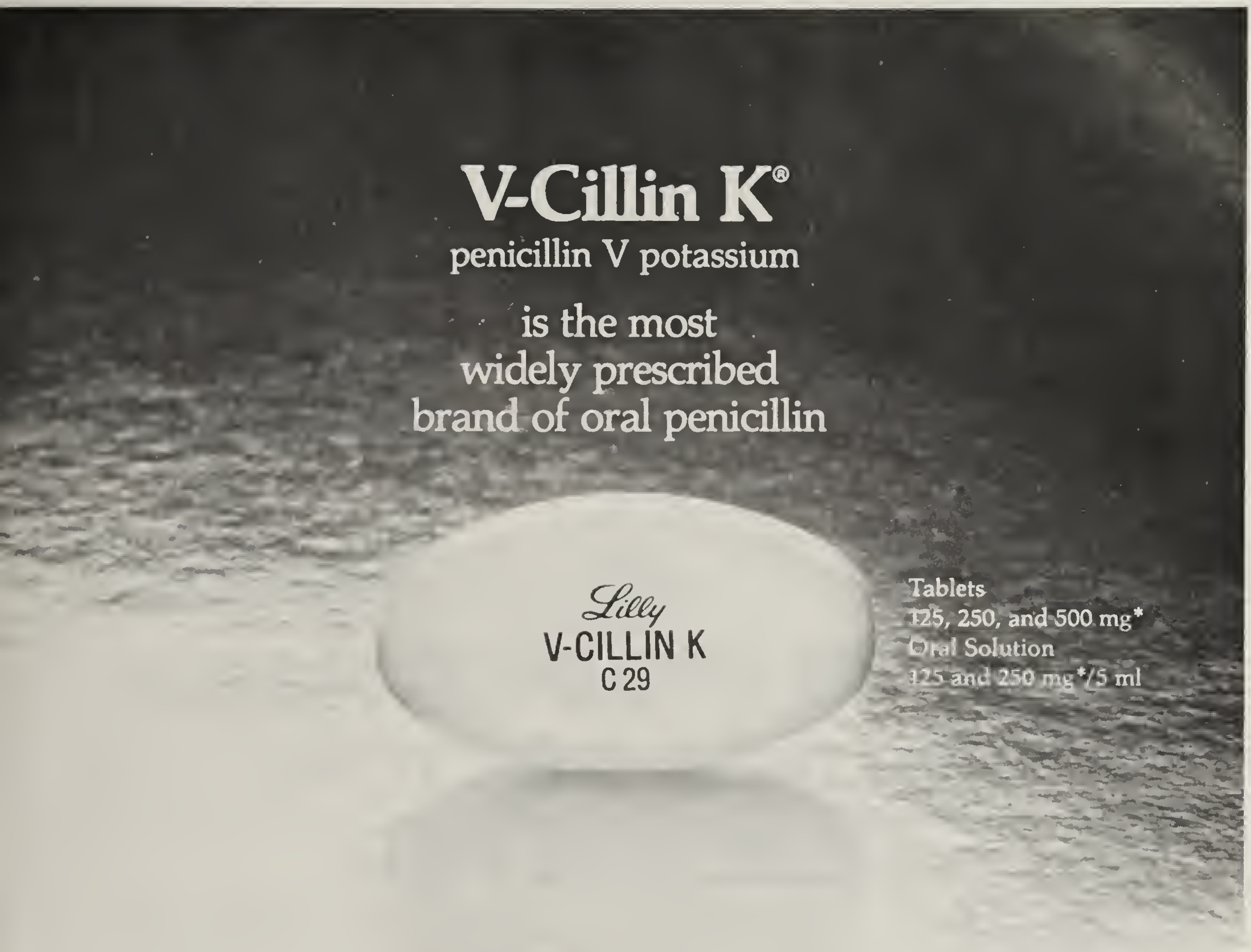

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The National Cancer Institute has developed a free "Helping Smokers Quit" kit to help you answer the "how" and to help your patients quit the smoking habit. Materials for this kit were pretested with the cooperation of the Harris County (Texas) Medical Society and M.D. Anderson Hospital and Tumor Institute in Houston, Texas.

The National Cancer Institute will provide the "Helping Smokers Quit" kit free of charge to all physicians who want to participate in this important effort. Included in the kit are guidelines for physicians, a self-test to help smoking patients determine why they smoke, pamphlets with tips on quitting, and waiting room posters to introduce the subject. Each kit contains enough materials to help 50 of your smoking patients who want to quit.

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THE CHICAGO DEPARTMENT OF HEALTH

NEWS LETTER

VOL. 18, NO. 1, MAY, 1978

AUTOMATIC BLOOD PRESSURE DEVICES

A Chicago Department of Health report on the Vita-Stat automated blood pressure machines concludes that their consistency and accuracy is sufficient for screening purposes.

The report, the first in the nation based on a scientific study by a public agency, was prepared under the direction of Dr. David M. Berkson, the Department's heart disease control officer. It consists of (1) a statistical evaluation by Drs. Shekelle and Raynor of Rush-Presbyterian St. Luke's Medical Center, based on data collected over a four month period by Lawrence Shireman and Thomas Whipple and their co-workers in the Health Department, and (2) an evaluation of the results with recommendations for regulatory actions if deemed necessary. The report notes at the outset that any assessment of automatic blood pressure devices must compare their measurements with measurements by trained observers recorded by the traditional cuff ausculatory method. Since blood pressure may vary rapidly, the assessment of accuracy of measurement is difficult.

"In our evaluation of the Vita-Stat machine," the report states, "we found that, when the machine and a trained observer measure blood pressure simultaneously, there is a very good agreement." It is essential, however, that a machine must perform well in sequential measurement with trained observers as well as in simultaneous measurement. In sequential measurement the machine, in general, has a fair degree of accuracy. It does not perform as well, however, as trained technicians. In the laboratory analysis of the comparison between one machine and several technicians the machine tended to read systolic pressure 3.4mm higher and diastolic pressure 2.3mm lower. In the field analysis the machine tended to read systolic blood pressure 1.5mm and diastolic pressure 0.5mm higher than the technicians.

Although agreement between technicians in mean blood pressure measurement and in categorizing persons as normal, borderline or hypertensive tends to be closer than technician-machine agreement, these differences are small and of little practical significance in screening.

The report recommends that if licensing is insituted by the City of Chicago, periodic recalibration and testing should be required. The public must be inforn that these instruments serve merely screening devices; that they do not repl medical consultation and that, beca blood pressure is subject to variati several elevations or lack of elevations not constitute a diagnosis of hypertens or normotension.

Blood pressure readings must interpreted by a qualified medical prof sional and self-adjustment of medicati based on automated blood pressure re ings without medical supervision may dangerous. The report concludes "t although the Vita-Stat machine does perform as well as trained technicians, consistency and accuracy is sufficient screening purposes."

Seminars in Chest Medicine

egionnaires' Disease:

resent Status

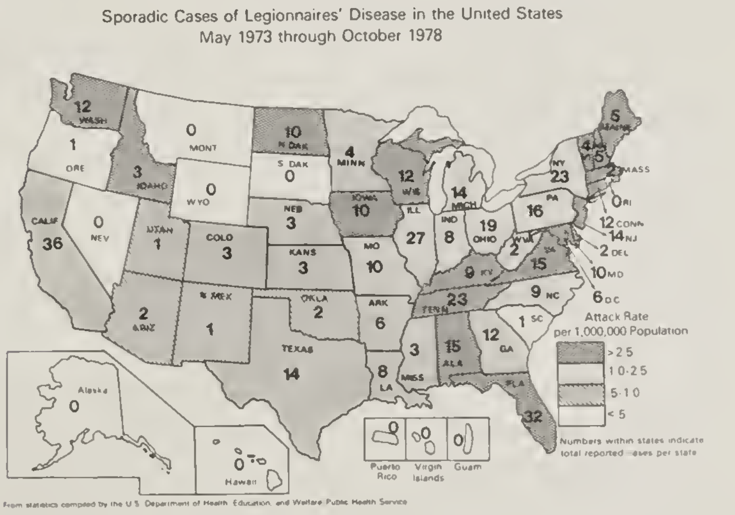
ark D. Garfield, M.D.

itors:
ysal M. Hasan, M.D.
chard I. Lemen, M.D.
rnard E. Levine, M.D.

In July, 1968 at least 144 persons at a county health cility in Pontiac, Michigan were affected by an acute, plosive, common-source outbreak febrile illness. The mptoms of fever, chills, headache and myalgia usually solved within 5 days without treatment. There were pneumonias and no deaths. A defective air-nditioning system was implicated as the source, but an iologic agent could not be determined.[18] Paired sera ere obtained and stored at the Center for Disease ontrol in Atlanta.

In July, 1976 at least 182 persons, attending an American Legion convention in Philadelphia, were affected by an explosive common-source illness defined as cough with temperature ≥ 38.9°C or as any fever with infiltrates on chest radiograph.[17] One hundred and forty-seven persons required hospitalization, 90% of the chest radiographs were abnormal, and 29 patients died. An additional 98 Legionnaire delegates not meeting the defined criteria reported fever, cough or chest radiography abnormality. By inoculating guinea pigs with lung tissue suspensions from 4 fatal cases, and subsequently inoculating embryonated hens' eggs with suspensions of tissue from the guinea pigs, McDade and co-workers were able to isolate a gram negative, non-acid fast bacillus.[7, 22]

Indirect fluorescent-antibody testing was performed by incubating yolk-sac cultures with serum specimens from patients meeting the clinical criteria for Legionnaires' disease. Fluorescent antihuman globulin antibody was then applied. Ninety-one percent (101 of 111) of the patients with suitably timed specimens showed diagnostic increases in antibody titers. Sixty-two patients seroconverted (fourfold increase in titer of convalescent compared to acute phase sera, with minimum ≥ 1:64), and 39 patients had single positive titers (minimum ≥ 1:128).[22] Stored sera from previously unsolved outbreaks of acute illness were also tested, with diagnostic results obtained in an epidemic of pneumonia in 1965 at a psychiatric hospital in Washington, D.C. and in the 1968 outbreak of febrile illness in Pontiac, Michigan.[7, 8, 22] Current concensus is that "Pontiac fever" was an outbreak of Legionnaires' disease, with the difference in pathogenicity related to dose response or host factors.[22] However, it should be noted that as of August, 1978, 17 strains of Legionnaires' disease bacteria (LDB) had been identified, the Togus strain, representing a new serogroup with only partial cross-reactivity to the other strains.[9] Therefore, the clinical manifestations of infection with LDB also may be secondary to variable pathogenicity of different strains.



Sporadic Cases of Legionnaires' Disease in the United States
May 1973 through October 1978

From statistics compiled by the U.S. Department of Health, Education, and Welfare Public Health Service

Figure 1

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om: University of Arizona Health Sciences Center, Division of espiratory Sciences, Tucson, AZ 85724.

Table 1
Frequency (%) of Symptoms in Patients Clinically Ill from Infection with LDB, Pontiac, Michigan Outbreak Compared to the Wadsworth Veterans Administration Hospital Outbreak.[19, 20]

Symptom	Employee Patients (n=95)	Visitor Patients (n=49)	Patients (n=24)
Fever	100	100	100
Malaise	98	96	100
Myalgia	95	96	100
Cough	57	24	100
Dry			45
Productive			55
Hemoptysis			38
Headache	91	84	25
Nausea	46	35	30
Chest Pain	45	24	42
Diarrhea	15	33	55
Preceding Fever			17
Concurrent Fever			38
Dizziness	54	47	
Confusion	23	10	

Epidemiology

Infection with LDB has been found to have both outbreak-associated and sporadic incidence. Sporadic cases have been reported in 43 states and the District of Columbia (see Figure 1). Outbreaks have been identified in Memphis, Dallas, New York City, Atlanta, Bloomington (Indiana), Los Angeles, Burlington (Vermont), Kingsport (Tennessee), Columbus (Ohio), Philadelphia, Pontiac (Michigan), Washington (D.C.), Nottingham (England), and Benidorm (Spain).[14, 16] The outbreak in Spain affected 164 (65%) of 252 tourists. Eighty-six (52%) developed respiratory symptoms and 3 died.[15] In the United States, all of the reported outbreaks (except the Wadsworth Veterans Administration Hospital) and the majority of the reported sporadic cases have occurred in the eastern half of the country. The prevalence of cases is highest during the late summer and fall.[16]

In 4 outbreaks (Bloomington, Memphis, Atlanta, and New York City) LDB has been isolated from the water of 3 cooling towers, 2 evaporative condensers, and one creek.[10, 11, 12, 16]This information has led to 3 hypotheses: the water and cases were both exposed to the same airborne source, the association is purely coincidental, or the organisms from the cooling systems were the source for the outbreak-associated cases. The third possibility is most favored. Investigations in Washington, D.C. and Benidorm, Spain have implicated an airborne route of spread, but not cooling systems.[12] There has been no evidence of person to person transmission.

Certain groups appear to have increased susceptibility for disease following exposure to LDB. Risk factors include male sex (3:1),[16] age > 60 (2:1),[17] smoking (3.4:1),[17] and immune-suppression. At the outbreak in Vermont, patients with malignancy, immunosuppressant therapy, or renal disese had a case fatality rate of 55% compared to 17% for patients without underlyi disease.[5]

Clinical Features

The manifestations of LDB infection range fr asymptomatic to life-threatening. The outbreak at t American Legion convention in Philadelphia w associated with a high incidence of pneumonia a mortality.[17] However, the 144 persons affected wi "Pontiac fever" did not have a single instance pneumonia or death;[18] and employees at t Legionnaires' convention hotel, despite serolog evidence of previous LDB infection, could not rec serious respiratory illness.[17]

Of those patients symptomatic from infection wi LDB, the published frequency of specific symptor varies (Table 1). A pattern of asymptomatic low gra fever progressing to maximum temperature ≥ 39.4 with associated weakness, malaise, anorexia, coug pleuritic chest pain and diarrhea has been emphasized highly suggestive for Legionnaires' disease hospitalized patients.[20] The incubation period estimated to be 24-48 hours.[18]

Of the patients requiring hospitalization during tl Philadelphia outbreak, greater than 90% had che radiograph abnormalities.[17] Infiltrates were multilob in approximately 75%[20] and were bilateral approximately 50%.[17, 20] The infiltrates frequent progressed to consolidation,[17] and small pleur effusions were not uncommon.[20]

Various laboratory abnormalities have been noted. moderate leukocytosis is present in most patients. addition, abnormal liver function test hypophosphatemia, hyponatremia, and abnorm urinalysis have been reported.[20, 21] It is unclear wheth these changes result from infection with LDB, represent pre-existing disease. The frequency of tl above laboratory abnormalities has not yet bee reported for the Philadelphia and Pontiac outbreaks.

Diagnostic Confirmation

The diagnosis of infection with LDB can be confirme by 3 methods: 1) Serology (indirect immunofluorescence).[7, 22] 2) Direct immunofluorescence. 3) Culture.[7, 19, 22] The methodology has alreay bee discussed, and is described in detail elsewhere, as cite above. Of the 453 sporadic cases reported, the diagnos was confirmed by serology alone 374, direc immunofluorescence alone 59, culture alone 4, serolog and direct immunofluorescence 8, culture and direc immunofluorescence 4, serology and culture 1, and all methods 1.[16] To date, Dieterle silver-impregnatio strain is the most sensitive method for demonstratin LDB in histologic sections. However, the specificity i not fully determined, therefore while highly suggestive the test is not considered diagnostic.[2]

The non-serologic methods have usually require tissue from open-lung biopsy or autopsy (52 of the 5 cases confirmed by direct immunofluorescence). T date, few positive results have been reported with les vasive methods (2 by lung aspirate, and 1 each by onchoscopy, thoracentesis, transtracheal aspirate, d sputum). Two cases were proved by culture of the eural fluid.[16]

eatment

In any patient with pneumonia of uncertain etiology, e differential should include Legionnaires' disease. sociated symptoms vary and are non-diagnostic; wever, a rapid increase in temperature to ≥ 39.4°C 02.5°F) and diarrhea should raise one's suspicions.

Confirmation in the acute setting remains difficult. ntil recently, histologic confirmation by direct munofluorescence usually required open lung biopsy, rely justified in non-immune-suppressed patients. owever, direct staining of sputum smears may be eful in some patients. By sampling multiple sputum mples, Broome, et al. established the diagnosis in 24% patients determined to have Legionnaires' disease by her criteria.[1] Indirect immunofluorescence, though n-invasive, may be falsely negative if the convalescent rum is obtained prior to 6 weeks after the onset of ness (In the Wadsworth study seroconversion was %, 64%, 82%, 91% and 100% at 2,3,4,5, and 6 weeks spectively).[20] Therefore, confirmation of gionnaires' disease is usually in retrospect, and in the sence of an epidemic often is a diagnosis of exclusion.

When Legionnaires' disease is suspected, or when ycoplasma pneumoniae is considered an alternate ssibility, erythromycin is an excellent drug of empiric e. If direct immunofluorescence is available, several utum specimens should be examined. Acute phase rum also should be obtained and stored frozen. Six eeks after the onset of illness, convalescent serum ould be obtained.

Recently the Center for Disease Control in Atlanta s offered to provide (without cost) the Legionnaire isease bacteria to any laboratory with the capability of direct fluorescent microscopy.[23] Therefore, the ysician can submit the paired sera to the nearest alified laboratory for diagnostic confirmation.

onclusion

Legionnaires' disease bacterium has been identified as e causative agent of both outbreak-associated and oradic disease. The manifestations of infection include nrecognized subclinical disease, self-limited febrile ness, pneumonia, and death. In hospitalized patients ith pneumonia of uncertain etiology, Legionnaires' isease should always be a consideration, but articularly in the setting of temperature ≥ 39.4°C, leuritic chest pain, and diarrhea. Serology is the mplest means to confirm the diagnosis, but is not elpful in the acute clinical setting. More rapid onfirmation by direct staining methods is possible in ss than 25% of cases. Therefore, in patients with neumonia of undetermined etiology, treatment with rythromycin 0.5 to 1.0 gram every 6 hours for a least 2 veeks should be considered.

References available upon request.

Dean's Clinical Rounds

Clinical Aspects of Pain

Part I

Stephen B. Shanfield, M.D.
Stuart R. Hameroff, M.D.
John T. Boyer, M.D.
Stephen S. Cox, M.D.

Pain is a universal human complaint. Acute pain is a warning system which alerts the individual that something is wrong and to seek relief, often from the medical care system. Chronic pain on the other hand has no biologic function and is frequent cause of disability. One report estimates that chronic pain syndromes cost $40 to $50 billion annually in hospital and health services, loss of work, litigation costs and compensation payments. There has been recent interest in chronic pain and a new system of care has grown up for the treatment of pain patients. The topic of pain has recently been the focus of intense discussion at a national level and goals for the next decade in relation to this topic have been identified.[1]

Thus the topic of pain is an important one for the Dean's Clinical Rounds. The discussants come from a wide range of backgrounds as is fitting in any discussion of pain. Dr. Stuart Hameroff is an anesthesiologist and head of the Pain Service at the Arizona Health Sciences Center. Dr. Stephen B. Shanfield is a psychiatrist at the Arizona Health Sciences Center who has written on pain. Dr. John Boyer is a rheumatologist at the Arizona Health Sciences Center and Dr. Stephen Cox is a physician with a special interest in geriatrics who is associate medical director of Hillhaven Hospice.

From: University of Arizona Health Sciences Center, Dept. of Psychiatry, Tucson, AZ 85724. Reprint requests to Dr. Shanfield.

Theoretical Mechanisms of Pain and Pain Control

Stuart R. Hameroff, M.D.

Patients in chronic pain tend to be depressed, dependent and have other psychological, emotional and legal problems making them difficult to manage. In the Pain Clinic at the University of Arizona Health Sciences Center we are pragmatic and utilize whatever therapies tend to succeed. Trigger points, trancutaneous nerve stimulation, antidepressants and psychological counseling are the stalwarts of our multidisciplinary treatment approach. Most experimental data about pain has been derived from acute pain animal models or human volunteers. Chronic pain is relatively poorly understood, yet has far greater medical and socioeconomic importance.

Chronic pain may apparently be imprinted or habituated by increased neuronal activity at various sites within the classical nociceptive pathways including peripheral receptors, spinal cord, thalamus or cortex.[2] The most significant new information related to pain and pain control is that certain other pathways modulate pain and that this modulation may be clinically useful.[3] Pain modulating regions include central gray matter adjacent to third vetricle and aqueduct of Sylvius. Reynolds[4] in 1968 first demonstrated that electrical stimulation of small areas of central gray caused marked behavioral analgesia in animals. Numerous studies in various species including man have verified stimulus provoked analgesia with maintenance of awareness, motor and sensory functions. The periaqueductal gray matter, a 4mm layer of fibers between third and fourth ventricles, receives diverse input from emotional, motivational and sensory systems. Axons descend from this region and synapse in the nucleus raphe magnus, a structure located below the floor of the fourth ventricle. From there, serotoninergic fibers descend in the dorsolateral funiculus and enter the dorsal horn at various levels of the spinal cord. Other raphe nuclei send ascending fibers to forebrain structures including hypothalamus and cortex. Pain impulses arriving in laminae 1, 2, and 5 of the dorsal horn and trigeminal nucleus are inhibited by these fibers, apparently via interneurons whose pain inhibitory neurotransmitter is enkephalin, an endogenous opioid or endorphin. This inhibition fulfills the tenets of the gate control theory proposed by Melzack and Wall in 1965.[5]

The endorphins, or endogenous opioids occupying receptors in common with morphine and other narcotics, and their analgesic activity are inhibited by the narcotic antagonist naloxone. Rates of endorphin activity and turnover are speculated to be involved in addiction problems and susceptibility to chronic pain. A Swedish study,[6] for example, measured endorphin levels in cerebrospinal fluid of a number of chronic pain patients. The data suggest that patients with truly severe pain due to well documented nerve injury tend to have low endorphin levels, while patients generally thought to be exaggerating their problems had normal to high endorphin levels.

The use of opiate antagonists have further implicat endogenous opioids in clinical stimulus provok analgesia. Several studies have shown acupunctu analgesia, for example, to be reversed by nalaxone.[7] Acupuncture is akin to trigger point injections a transcutaneous electrical nerve stimulation which a clinically useful in treatment of chronic pain. Trigg points, usually located by eliciting areas of extre tenderness and paresthesia, are in consistent anato positions among patients. The paresthesia, as well chronic pain distribution often follow non dermato pathways which mimic acupuncture meridians. The may be related to diverse, small unmyelinat autonomic fibers. Melzack has corroborated the clinic impression that trigger points are anatomically identic with acupuncture points.[9] Early observations on trigg point therapy indicate that analgesia was obtain regardless of the mode of stimulation.[10] Loc anesthetics, saline, dry needling, or electric stimulation at specific loci may apparently activa central endorphin analgesic mechanisms which may al mediate placebo reactions.

Depression commonly accompanies chronic pai Tricyclic antidepressants are beneficial in many chron pain conditions. The relation between mood and clinic tolerance to chronic pain is complex. Recent data fro our laboratory suggest that endorphins mediate som aspects of tricyclic induced therapeutic respnse chronic pain.

Chronic pain patients are often viewed as a anathema by physicians because of their dependenc problems and psychological difficulties. We now have least a glimmer of understanding of the mechanism involved and realize that chronic pain can be cause psychological problems as well as a result. Therefore, w attempt to help as many patients as possible utilizing multidisciplinary approach.

Psychiatric Aspects of Chronic Pain

Stephen B. Shanfield, M.D.

Pain is a complex phenomena which has multip meanings. It commonly stands for three related b separate phenomena; impending or actual tissue dama due to a peripheral stimulus, pain behavior or respons and a subjective experience.[1]

Impending or actual tissue damage causes acute pa and has an important biologic function. The pain war the individual that something is wrong, and is also signal to the physician to look for the etiology of th problem.[1] Some pain, such as cancer pain, would be this category even though it may last for a long tim Treatment is aimed at alleviating the pathologic proce and analgesia is the mainstay of treatment.

Behaviors associated with pain like grimacin wincing and moaning are often symptoms signaling a acute disease process. However, these behaviors can b present in the absence of any disease process as is ofte true in chronic pain. Many chronic pain patients hav little or no demonstrable tissue pathology yet have behaviors which communicate pain. Often the behaviors are maladaptive ways to deal with emotional problems, developmental crises or other life stresses. The focus is thus diverted from the crisis to the pain and its behaviors, and problems become subsumed under the problem of pain. Treatment is aimed at identifying and dealing with the emotional difficulties and changing the pain behavior.12

Pain is also a personal subjective experience. In this sense all pain from whatever source has a psychologic and feeling component. Because of pain's subjective nature it is difficult to objectify for both clinical and research purposes. The way pain is experienced and expressed is dependent on the disease process, the personality style, the culture and the present life circumstances. It is well known that the pain experience is modified by emotional states such as anxiety or depression.[13] Present circumstances are quite potent in modifying pain as in the well known stories of the disregard for the pain of a fresh wound in the heat of battle.

Pain is seen commonly in transient situational stress n which psychic suffering is converted into pain. In addition, under stress, momentary lapses of attention can result in accidents which cause pain. That these experiences are common is witnessed bv the enormous over-the-counter sale of non-prescription analgesics.

The pain threshold is decreased by anxiety. A supportive relationship can diminish anxiety and decrease pain. The study of taking patients through the operating room pre-operatively and decreasing the need for analgesics post-operatively, points to the value of support in diminishing anxiety and pain.

Depression decreases the pain threshold so that pain may be made worse. The depressive syndrome is very common, particularly in the elderly, and pain is one of the more common depressive symptoms.[14] Indeed, every specialist has patients with pain of depressive origin. Some patients refer the pain to the eyes, some to the ears, and some speak directly of psychologic suffering. Also patients with pain from a peripheral source, over a long period of time, may become depressed. It is important to treat the depression with support, efforts at conflict resolution and where appropriate, antidepressant medication. Often the pain will diminish as a result of this treatment. In chronic pain the behaviors also have to be modified in addition to the treatment of the depression.[12]

It is often necessary to engage the family in the treatment of the patient with chronic pain since the family can reinforce pain behavior. Frequently the patient's suffering is experienced intensely by the spouse who also has increased psychologic symptoms which require treatment.[15]

For those few who can be hypnotized deeply, the pain experience can be changed directly. However for most, the pain experience is modified indirectly by hypnosis, by shifting the focus of the patient away from the pain. Biofeedback and relaxation exercises are also helpful in shifting the focus of the patient and relieving stress and pain.

Often the chronic pain patient can be given work at just below the pain threshold and work can be increased slowly. Here the pain threshold is slowly increased and the patient increases his productivity.

The chronic pain patient may be difficult to deal with. The patient is skeptical of treatment outcome in spite of his therapy seeking behavior. The physician can be led to inappropriate searches for cure that result in frustration for both patient and doctor. The patient may abuse and be taking many medications. Litigation often serves as a reinforcer of the pain and can frustrate treatment. Because of the intractability of complaints, hostility and rejection may be evoked. Indeed not all physicians are suited temperamentally to work with these patients. A hopeful, supportive and non-judgemental posture associated with realism and firmness is quite helpful in dealing with the chronic pain patient.

The treatment of the patient with chronic pain should be multidisciplinary, with indviduals from many specialties as members of the team, including anesthesiology, neurology, neurosurgery, occupational therapy, pharmacy, psychiatry, physiatry, psychology and rehabiliatation. Often the patient may need intensive treatment over weeks, either as a day patient or over-night patient in a specialized pain treatment center. Crucial in dealing with patients with pain is the early diagnosis and the treatment of a potentially chronic condition.

To be continued

Seminars in Endocrinology
and Metabolism

Hyperthyroidism Possibly Induced by Iodine in a Multivitamin

Marshall B. Block, M.D.
Salvatore J. DeFrancesco, M.D.

Editor:
Stephen E. Dippe, M.D.

Introduction

Increasing recognition of atypical varieties of hyperthyroidism has led to greater scrutiny of patients who present with elevated peripheral thyroid hormone levels.[1] Iodine-induced hyperthyroidism is one such condition which can be misdiagnosed unless careful attention is paid to the patient's history and appropriate laboratory studies are obtained. We recently had an occasion to study a postpartum woman who presented with the classic signs and symptoms of hyperthroidism but who had a very low radioactive iodine uptake. Careful history and further diagnostic studies over several months raised the question of iodine induction secondary to a multivitamin (Natalins). The subtle source of iodine and its widespread use prompted this report in order to draw attention to it and uncover other similar circumstances which have been unreported.

Case Presentation:

A 26-year-old woman was noted to have an enlarged thyroid gland during routine evaluation. It was approximately 30 grams in size and peripheral thyroid hormone levels were normal. Eight months later she became pregnant, but no further enlargement of her thyroid gland was noted. However, following delivery the patient developed nervousness, irritability and weight loss and peripheral thyroid hormone levels were again measured and found to be elevated (T_4 by RIA = 25.0 [4.5-ll.5 ug./dl.], T_3-RU = 44% [25-35], T_7 = 11.0 [1.1-4.0], T_3 by RIA = 635 ug./ml. [56-180]). Clinically she appeared to be tense, nervous, with a history compatible with hyperthyroidism. On physical examination exophthalmos was absent but lid retraction and lid lag were present. There was a fine tremor to her outstretched hands and skin temperature was warm. Reflexes were rapid and on palpation of her neck an approximatley 35 gram thyroid gland was felt. Both the right and left lobes were diffusely enlarged without any nodules palpated. No lymph adenopathy was felt in the neck and the gland moved freely with deglutition. No bruits were heard over the goiter. An I_{123},radioactive iodine uptake was 0% at 6 and 24 hours. A 24 hour urinary iodine level was 700ug. A careful drug history was then obtained and the only medication that the patient had been taking during the last eight months of her pregnancy were Natalins, a multivitamin preparation manufactured by Mead-Johnson. Discontinuance of the Natalin preparation and conservative management with Inderal 10 mg. four times a day was undertaken.

Approximately six weeks later a repeat 24 hour radioactive iodine uptake was 11% and the scan revealed irregular uptake within both lobes of the thyroid gland. Peripheral thyroid hormone levels were still elevated but lower than initial values and management with Inderal was continued.

Two months later she noted lethargy and had gained six pounds. Reflexes were sluggish and examination of her neck revealed an approximately 40 to 50 gram, diffusely enlarged, lympho-nodular-feeling thyroid gland. No bruits were heard over the goiter. Peripheral thyroid hormone levels were now below normal (T_4 RIA 3.4 mcg.%, T_3 RU 21.2%, $T_7$0.72, T_3RIA 68 ng./dl) and serum TSH was 23.7 mIU./ml. (normal < 5.0 mIu./ml). Thyroid antibody titers were obtained and revealed a negative thyroglobulin level but a microsomal titer of 1:6400 (normal < 1:64 for both.)

The patient was started on thyroxine relpacement 0.15 mg. per day and Inderal was discontinued.

Discussion:

Our patient initially presented with a slight enlargement of her thyroid gland of uncertain etiology and subsequently was given a multivitamin preparation (Natalins), commonly used during pregnancy. She subsequently (seven months later) developed symptoms and signs and biochemical evidence for hyperthyroidism. Because of low I_{123}uptake and high urinary iodine levels, iodine-induced hyperthyroidism was suspected. Recovery of thyroid gland function several weeks after withdrawal of the offending agent

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From: Departments of Medicine and Obstetrics, St. Joseph's Hospital and Medical Center, Phoenix, AZ 85013. Reprint requests to M. B. Block, M.D., 3411 North 5th Ave., Phoenix, AZ 85013.

seemed to further support this contention. The patient had underlying Hasimoto's thyroiditis which was supported by positive microsomal antibody titer and the development subsequently of primary hypothyroidism.

The sequence of events appears unique for previous studies in patients with chronic lymphocytic thyroiditis have shown an increased susceptibility to the development of myxedema during iodine administration.[2] However, large doses of iodine (up to 180 mg. per day) were utilized in these studies. Although pre-existing iodine deficiency was thought to be a prerequisite for the development of hypothroidism previous studies have demonstrated that this is not necessary.[3] Furthermore the high daily intake of iodine in the southwest United States in addition to the content of iodine within the multivitamin preparation[4] may have been sufficient to produce hyperthyroidism. Low doses of iodine in patients with chronic lymphocytic thyroiditis have not been systematically studied and thus it is quite possible that the cause of our patient's hyperthyroidism may be more common than is appreciated.

The multivitamin preparation Natalins, manufactured by Mead-Johnson, is listed as containing 150 mcg. iodine per dose.[5] However, a survey of the iodine content of various pharmaceuticals revealed a microgram iodine content per dosage of 375 for Ntalins.[6] Thus there probably is a greater content of iodine in the capsules than is appreciated even by the manufacturer.

The recent recognition of variants of hyperthyroidism associated with low uptake of radioactive iodine also needs to be considered in the differential diagnosis of this patient's condition. However, the elevated urinary iodine level is not compatible with the natural historv of that disorder and it was therefore excluded from consideration.

In summary, an unusual presentation of hyperthyroidism possible secondary to iodine ingestion in the form of multivitamin preparation, Natalins, is presented in a patient who had underlying Hasimoto's thyroiditis. The unique features of this patient include the low dose and source of the iodine. Careful attention to underlying thyroid abnormalities in the administration of pharmaceuticals which contain iodine should always be borne in mind by those caring for patients with thyroid disorders.

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Seminars in Gastroenterology and Liver Disease

Proctalgia Fugax

A Pain in the Rear

George E. Burdick, M.D.

Editors:
Leon Rigberg, M.D.
Stephen Glouberman, M.D.
George Burdick, M.D.

Proctalgia fugax is a condition characterized by irregular, intermittent, severe pain in the rectal area. The etiology of this disorder is not known. Most often the attacks occur in young men (although women are not excluded) and are precipitated by a number of factors including sexual intercourse, explosive bowel movements, prolonged sitting particularly on a cold object, and nervousness. The attacks of pain are very severe. Fortunately, the episodes are usually short lived. Characteristically, individuals are awakened from a sound sleep with agonizing pain in the rectal region although the attacks may occur at any time including while excercising.

Despite the severe pain produced by proctalgia fugax these patients do not have definable disease of the rectum, colon or prostate. I have seen a large number of these patients over the years and have never been able to demonstrate significant disease on sigmoidoscopy or barium enema. Patients with this syndrome rarely have bowel complaints except during the attacks when some patients note an urgency to have a bowel movement due to the rectal spasm.

From: 1010 E. McDowell, Ste 204, Phoenix, AZ 85006

Present theory would incriminate spasm of the striated muscles of the pelvic floor as the cause of the severe pain. This is based on many clinical observations including rectal examination done by patients during attacks (the disorder seems uncommonly frequent in physicians). The anal tone has consistently been noted as normal but bands of spastic tissue interpreted as representing either levator ani or pubococcygeus muscles are often described just inside the anus. Additionally, amyl nitrate has no effect on the attack adding to the suspicion that the muscular spasm is due to contraction of striated muscle rather than smooth muscle.

A number of factors have led to under-recognition of the frequency of this condition. These factors include: the rather short duration of attacks; the relative infrequency of attacks, the fact that serious associated disease is rare; and many patients are ashamed of the symptomatology and are reluctant to complain. The major need for diagnosis is reassurance to the patient and to avoid excessive evaluation in search of exotic disease.

The attacks are frequently too brief to advocate the use of analgesics. One physician I have treated for this condition did take a codeine tablet during an attack which he thought may have increased the duration of that particular episode and did not help his pain. The most consistent form of treatment is the application of heat. This is most effectively accomplished by warm sitz baths which usually produces very prompt relief of discomfort. Upward pressure on the rectum has also been advocated by some. A surgical approach to the disease would seem to be risky and unnecessary.

Obstetrics and Gynecology

"Wrongful Life"-An Obstetrical Dilemma

Donald J. Ziehm, M.D.

Donald J. Ziehm, M.D., Editor

During recent years, in this country we have all been painfully aware of the sharp increase in malpractice litigation and the escalation in awards given to plaintifs. As a result malpractice insurance has become progressively more costly and difficult to obtain, especially for physicians in surgically oriented specialties. The problems have produced a crisis in medicine, affecting doctor-patient relationships, medical evaluation and decision making, and in some cases, the very ability to continue in practice. Many knowledgeable individuals have wondered whether the practice of medicine, as we know it, will be profoundly and perhaps permanently altered. Obstetrics and gynecology have obviously not escaped these difficulties.[1]

Although most birth defects are completely unpredictable some potential problems can be indentified. The risk to a fetus following first trimester maternal Rubella is well known. In addition, recent advances in intra-uterine diagnosis have made it possible to detect a great many genetic conditions. Only recently have the courts begun assigning direct responsibility to the obstetrician to inform the prospective parents of known risks, and are expecting prenatal diagnostic techniques to be employed where applicable.

Reprinted by permission from *Malpractice Lifeline* (Vol. 4, #1, Jan. 29, 1979), the next two issues will contain a review of the current status of recent court decisions concerning these crucial problems.

DJZ Editor

From: 1718 W. Glendale Ave, Phoenix, AZ 85021

Wrongful Life Litigation: The Debate over Damages

Rapid developments in antenatal diagnosis of genetic eases may have spawned a new set of medical ilpractice problems. What is the duty of the physician recognize and to warn prospective parents that their us may be afflicted with a severe, incurable disease? oes the infant born with a severe genetic disease have a ht to sue the doctor for failing to warn its parents? If ere is a cause of action against the doctor, for what mages is he liable? Over the past four years judicial swers to these questions have been changing in favor the plaintiffs.

The first major case on this subject of *Gleitman v. sgrove*, 227 A. 2d 689, decided by the Supreme Court of w Jersey in 1967. That court refused to permit a child th congenital defects caused by rubella or his parents sue physicians for failing to advise the prospective rents of the risk of birth defects so that they could ort the fetus. Essentially, the plaintiffs argued that the ant would be better off not to have been born. In ply, the chief Justice wrote "to recognize a right not to born is to enter an area in which no one could find eir way."

There the matter rested until 1975 when the Texas preme court decided *Jacobs v. Theimer,* 519 SW sd 846. e Jacobs case was also brought by a family with an fant deformed by rubella. Breaking sharply with the eitman precedent, the Texas court ruled that the ctor who knew that the pregnant woman had been posed to rubella had failed to satisfy a duty to disclose e risks that the virus posed to the fetus. Refusing to t involved in trying to measure the difference between n-existence and a defective existence, the court ruled at the parents had a valid cause of action against the ctor to recover as damages the money expended for e care and treatment of the child's physical pairment.

During the past three years lawsuits brought by rents against doctors after the birth of children with netic disease have begun to appear. Although it is too rly to predict what the ultimate rule of law will be, a end is emerging. First, as in Gleitman case, the courts efer to deal with the parents as plaintiffs, rather than e infant. Second, the courts seem quite willing to hold ysicians responsible in damages for medical costs ising out of their failure to alert pregnant women to sks of bearing children with certain well know genetic seases. Third, the courts are hesitant to award other nds of damages, such as for emotional pain and ffering. This is important because juries often make ry large awards on this count.

Recently, a Federal District Court in Pennsylvania ecided the case of *Gildiner v. Thomas Jefferson University ospital* 451 F. Supp. 692, which typifies recent litigation. hortly after she became pregnant in 1973, Linda ildiner learned that she and her husband were carriers of the gene for Tay-Sachs disease. Knowing that her fetus was at a one-in-four risk for this disorder, the woman underwent amniocentesis. The test results indicated that the fetus was not affected, and she continued the pregnancy. Sadly, she gave birth to a son who was affected.

Charging that the amniocentesis was performed negligently, the parents and the infant sued the hospital and individual doctors. The defendants moved for judgement on the pleadings, arguing that no plaintiff had stated a claim for which relief would be granted. As to the infant plaintiff, the court agreed with the defendants. Citing the Gleitman case, the judge ruled that the infant with Tay-Sachs disese who would have been aborted if the disease had been diagnosed in utero "had not sustained damages cognizable at law."

However, the court thought differently of the claim filed by the parents, holding that "the public policy of Pennsylvania supports the recognition of a cause of action for damages arising from negligence in the performance of testing for Tay-Sachs disease." Finally, the court addressed the question of damages. It held that the parents would be entitled to recover damages for the medical treatment of their son. Unfortunately, the court refused to decide what, if any, other damages could be awarded at trial if the defendants were found to be negligent. This is the next question to be resolved in the "wrongful life" law suit.

New York Court of Appeals Rules on Legality of Suing Doctors For Birth Defects

Further support for legal editor Phillip Reily's conclusions about wrongful life suits came from the New York Court of Appeals in Albany the last week in December. In two separate cases, Long Island couples sought to determine whether parents of children born with birth defects can sue physicians for damages-both for the costs of raising children they might not have had if they had been properly warned by the doctors of the possibility of defects, and on behalf of the defective children themselves.

In 5-2 rulings the court said parents can sue their doctors to try to recover costs of raising such children, but rejected the proposal that parents be allowed to collect extra compensatory damages in the names of the children. In one case, a woman who became pregnant at 37 and her husband sued three physicians for allegedly failing to advise her that she might bear a child with Down's Syndrome, an increased possibility for women her age. The second case involved a suit agains two physicians brought by a couple who say the were misled by their doctors about the prospects that their second child might inherit polycystic kidney disease. With the legal way now cleared on one issue, both cases now must be decided in separate trials.

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Pediatrics

Infant Botulism:

A Review of Three Phoenix Cases

David R. Alexander, M.D.
David R. Lersch, M.D.
Albert P. Pepka, M.D.
Allen M. Kaplan, M.D.

Editor:
Melvin Cohen, M.D.

Introduction

Botulism is a well-known illness usually caused by the consumption of food products containing the neurotoxin produced by *Clostridium botulinum*. Infants in the past had been felt to be immune from this disease as their specialized diets would not contain foods contaminated by the toxin. However, it is now known that an infant form of botulism can be caused by ingestion of *C. botulinum* spores or vegetative cells. Botulinal toxin is produced in vivo after multiplication and lysis of the bacterium in the infant's gastrointestinal tract. The developing signs of neuromuscular paralysis are similar to those of the adult form of botulism and result in an illness varying from inapparent intoxicati to a fulminating fatal disease. The purpose of this repc is to review three cases of botulism occurring in infar in Phoenix. A detailed clinical review of infant botulis will be presented.

Case Reports

Case 1. A 6-week-old infant was admitted to Joseph's Hospital with a two-day history of increasi lethargy and poor feeding. The infant was the product a normal pregnancy and delivery, and the neona period was uneventful. The infant was started Similac but later changed to a soy formula because irritability. There was a one-month history intermittent fevers, and the infant completed a course antibiotics for otitis media two days prior to admissic There was no history of constipation. The family lived an outlying commmunity where the city water supp was stored in a tank on the mountainside. The tank w allegedly condemned as unsafe due to excessi sediment deposits. (Culture of the sediment and ass for botulinal toxin was negative.)

Physical examination on admission revealed hypotonic infant with diffuse muscular weakness an poor sucking reflex. The cardiopulmonary a abdominal exams were normal. Neurologic examinati revealed intact extraocular movements and pupilla responses. There was evidence of bilateral fac weakness and absence of gag and corneal reflex Response to painful stimuli was minimal, and de tendon reflexes were reported to be symmetrical a brisk.

Laboratory studies including a complete blood cou serum electrolytes and chemistries, and thyrc function studies were normal. A lumbar punctu revealed a clear cerebrospinal fluid (CSF) with no ce and a normal glucose and protein. Toxic and metabo screens were negative. An electromyogram (EM showed small amplitude and low-voltage motor u action potentials consistent with botulism. Cultures the blood, urine, and CSF revealed no growth. Seru and stool specimens for botulinal toxin and cultures *Clostridium botulinum* were sent to the Center for Disea Control (CDC) in Atlanta, Georgia (Table 1).

The hospital course was characterized by intermitte constipation but gradual neuromuscular improveme Antibiotics including ampicillin (200 mg/kg/day) a gentamicin (7.5 mg/kg/day) were started a discontinued when cultures were negative. The infa was discharged after an 18-day hospitalization. Follo up examination revealed a normal infant.

Case 2. A 3-week-old infant male was admitted Desert Samaritan Hospital with a two-day history weakness, lethargy, and poor feeding. Intermitte apnea and bradycardia were noted, and the infant w transferred to Good Samaritan Hospital for furth evaluation and treatment. The infant was the product a normal pregnancy and delivery by Cesarean section f cephalopelvic disproportion. The patient had minin

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From: Departments of Pediatrics and Pediatric Neurology, Good Samaritan Hospital, Phoenix, AZ 85006. Reprint requests to Dr. Kaplan, Department of Pediatric Neurology, Good Samaritan Hospital, 1033 East McDowell Rd., Phoenix, AZ 85006.

Table 1
Botulinal Toxin Studies

Case	Toxigenic Type	Fecal Specimen	Blood Specimen
1	B	+	-
2	A	+	-
3	B	+	+

respiratory difficulties at birth, but there were no further neonatal complications. The infant was breast fed exclusively, and there was no history of constipation.

Physical examination on admission revealed a weak and lethargic infant with pooling of oral secretions in the hypopharynx. The infant had a poor sucking reflex, and weak cry, and gag responses were hypoactive. Extraocular movements were intact, but pupillary responses were sluggish. Anal sphincter tone was decreased. The infant was generally hypotonic with facial weakness. Deep tendon reflexes were brisk.

Laboratory studies including a complete blood count, serum electrolytes and chemistries, and urinalysis were normal. A lumbar puncture revealed a clear CSF with two white blood cells (lymphocytes). The CSF glucose and total protein were normal. Cultures of the blood, urine, and CSF revealed no growth. Initial blood gas studies were normal. An EMG early in the course was interpreted to be normal, but a repeat EMG revealed abundant small amplitude and short duration action potentials consistent with infantile botulism. Chest roentgenograms suggested a small aspiration pneumonia. A computerized tomographic scan of the cranium was normal. Serum and stool specimens for botulinal toxin and cultures for *C. botulinum* were sent to the CDC (Table 1).

The hospital course was characterized initially by progressive weakness with hypoventilation, carbon dioxide retention, and loss of deep tendon reflexes. Antibiotics including ampicillin (200 mg/kg/day) and gentamicin (7.5 mg/kg/day) were started pending culture results. A Tensilon test was negative, and repeat lumbar puncture again revealed normal findings. The infant never required assisted ventilation. There was gradual improvement of the neuromuscular process after the first week, and the infant was discharged after an 18-day hospitalization. There were no neurologic residua.

Case 3. A 6-week-old male infant was admitted to Desert Samaritan Hospital with a 48-hour history of lethargy and poor feeding. The infant had been seen the day prior to admission and was given Mycostatin for oral thrush. On the day of admission the mother noted a poor sucking reflex with increased pooling of secretions. The infant was subsequently transferred to St. Joseph's Hospital for further evaluation. The infant was the product of an uncomplicated pregnancy and delivery, and there were no neonatal complications. The infant was born in California, but the family moved to Mesa, Arizona when the infant was two days old. The mother related history of constipation since birth with the infant having only four stools by six weeks of age.

The infant had been breast fed primarily but received apple and prune juices for constipation. (Culture for *C. botulinum* and assay for botulinal toxin was negative.)

Physical examination on admission revealed a severely hypotonic infant with a poor sucking response, weak cry, and pooling of oral secretions in the hypopharynx. The infant was periodically apneic and required intubation and assisted ventilation. Cariopulmonary exam was otherwise normal. Neurologic examination revealed generalized muscular weakness with evidence of facial diplegia and external ophthalmoplegia. Gag, pupillary, and corneal reflexes were sluggish. Anal sphincter tone was diminished, and deep tendon reflexes were hypoactive.

Laboratory studies including a complete blood count, serum electrolytes and chemistries, and urinalysis were normal. Cultures of the blood and stool revealed no gowth. An EMG revealed brief duration low amplitude action potentials consistent with botulism. Serum and stool specimens for botulinal toxin and cultures for *C. botulinum* were sent to the CDC (Table 1).

The hospital course was characterized by persistent flaccid paralysis with bulbar weakness. The infant required assisted ventilation for nearly two months. Hyperalimentation and/or nasogastric feedings were necessary throughout the hospital course. Intermittent constipation also persisted. Ampicillin (200 mg/kg/day) and gentamicin (7.5 mgk/kg/day) was started but discontinued. Gradual improvement of the neuromuscular signs occurred with increasing extraocular movements and return of pupillary and deep tendon reflexes. The infant was discharged after a two and one-half month hospitalization. Follow-up three months after discharge reveals mild residual weakness. The infant is otherwise normal.

Discussion

Infant botulism was first recognized as a distinct clinical entity by Pickett et al[1] in 1976. Since this initial description, over 100 cases of infant botulism have been reported to the Center for Disease Control in Atlanta, Georgia. At least 45 of thee cases were reported from California and based on these statistics, approximately 250 cases of infant botulism would be expected to occur annually in the United States. The disease, however, has now been reported from three continents: North America, Europe (England),[2] and Australia[3] but as our knowledge, awareness, and ability to recognize this disease improves, diagnosis of infant botulism may well increase.

The *Clostridium botulinum* organism is a gram-positive anaerobic bacillus which produces heat-resistant spores. Seven toxigenic types have been recognized, but four types (A, B, E, and F) are known to cause disease in man. The organism is ubiquitous and is widely distributed in soil, dust, marine sediments, and raw foods. Under the

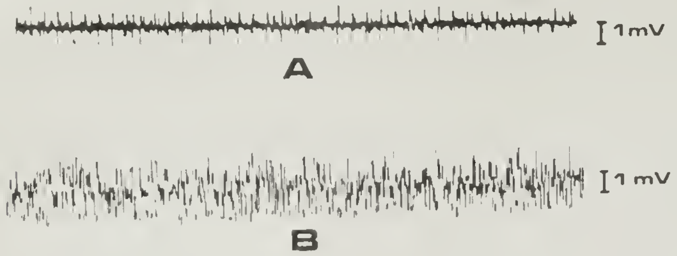

Figure1
Diagrammatic electromyograms comparing small amplitude, brief duration action potenials of infant botulism (A) and a normal interference pattern (B).

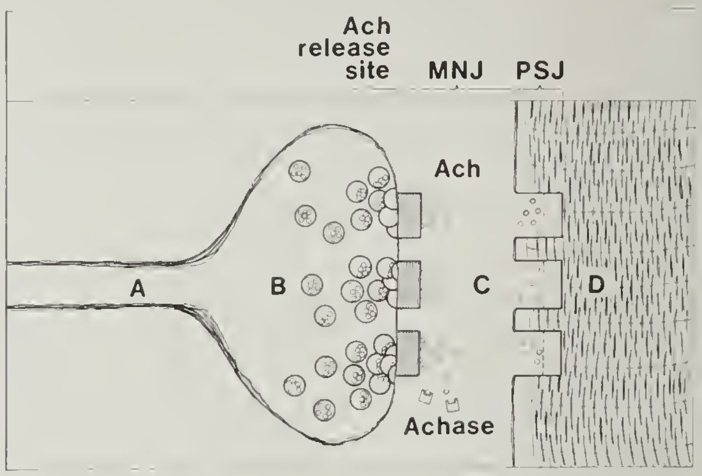

Figure 2
Myoneural junction illustration differentiating the site o pathophysiologic abnormalities in : A -Guillain-Barre-Stroh syndrome, B - botulism, snake and black widow toxins, C organophosphate intoxication with acetylocholinesterase block, an D - myasthenia gravis. Abbreviations: MNJ Myoneural junctio PSJ Post-synaptic junction, Ach Acetylcholine, Achase Acetylocholinesterase

appropriate conditions of an anaerobic environment (not always necessary), decreased acidity (pH greater than 4.6), and optimal temperatures of approximately 30°C (86°F), spores germinate and produce a heat-labile toxin. The toxin has been described as one of the most potent poisons known and based on studies of relative toxicity, the lethal dose for man has been calculated to be as low as 10^{-9}mg/kg of body weight.[4]

The clinical features of infant botulism, as our cases demonstrate, form a distinct constellation of signs and symptoms (Table 2). The clincial course ranges from mild symptomatology to severe, overwhelming neurologic dysfunction. Infants are usually healthy until onset of the disease between 3 and 20 weeks of age. The sudden presentation of generalized muscle weakness, lethargy, poor feeding and crying, and pooling of oral secretions is characteristic. Physical findings include ptosis, external ophthalmoplegia associated with sluggish pupillary light responses, facial weakness, diminished gag reflexes, and poor anal sphincter tone. The deep tendon reflexes may be normal initially but usually become hypoactive to absent later. Apnea and acute respiratory failure are not uncommon, and patients may require assisted ventilation for prolonged periods of time (Case 3). Constipation secondary t intestinal hypomobility is a prominent part of the syndrome and may present long before the onset of neurologic dysfunction (Case 3). This symptom, however, may be so mild as to go unrecognized. Electromyography may be helpful in supporting the diagnosis of infant botulism. A characteristic EMG pattern consisting of brief duration, small amplitude, overly-abundant motor unit action potentials (BSAP) has been described (Figure 1).[5] The EMG abnormality may persist until clinical recovery from the disease occurs.

Table 2
Review of Clinical Features

Symptoms/Signs	Case 1	Case 2	Case 3
Symptoms			
Muscle weakness	+	+	+
Lethargy	+	+	+
Poor feeding/sucking reflex	+	+	+
Weak cry	-	+	+
Pooling of secretions	+	+	+
Constipation	+	-	+
Signs			
External ophthalmoplegia	-	-	+
Pupillary abnormalities	-	+	+
Facial Weakness	+	+	+
Absent gag reflex	+	+	+
Hypotonia	+	+	+
Poor anal-sphincter tone	-	+	+
Apnea	-	+	+
Respiratory failure	-	-	+
Hypoactive reflexes:			
Early	-	-	+
Late	+	+	+

A recent report by Arnon et al[4] suggests that infant botulism may be a cause of the sudden infant death syndrome (SIDS). Fecal specimens from 211 SIDS cases revealed *C. botulinum* or evidence of toxin in 4.5% of cases. This recovery percentage may be falsely low due to the suboptimal samples obtained from many of the postmortem cases. Although the two diseases appear to have different presentation with botulism representing a progressive disease over several days and SIDS a more dramatic course in a previously healthy infant, the potency of botulinal neurotoxin presents the possibility that SIDS may represent a fulminant form of infant botulism. The authors feel further investigations will be necessary to clarify this association.

The pathogenesis of infant botulism differs remarkably from that of the adult form of the disease. The adult variety results solely from the ingestion of preformed toxin, the so-called "toxin-ingestion" theory. The pathophysiology of the infant form, however, is based on the ingestion of *C. botulinum* spores or vegetative cells with colonization and later production of the toxin—"toxin-infection" theory. The precise location of the effect of botulinal toxin on the peripheral

romuscular junction was demonstrated by Brooks in
3.[6] The toxin interferes with nerve conduction
ximal to the site of acetylcholine (ACh) release.
erefore, the ACh release mechanism is inactivated
irectly by failure of the impulses to reach the site of
mation and release of this neurotransmitter.
hough the source of *C. botulinum* organisms in the
nt variety remains obscure, contamination sources
e been found in six cases.[7] These included dust (one
e), soil (one case), and ingestion of honey (four cases).
erestingly, 13 of 43 (30.2%) of the California cases of
nt botulism were fed honey prior to the onset of
nptoms. In four of these infants, *C. botulinum* was
ated from the honey samples.[8] Because of these
dings and further studies on the "Honey
nnection,"[9,10] the Sioux Honey Association and CDC
ve recommended that honey not be fed to infants
der one year of age. The disease, however, has
urred in exclusively breast-fed infants, and the
portance of environmental or host susceptibility risk
tors cannot be overlooked. Studies are currently in
gress investigating the possible role of maternal
tibodies, gastric acidity, gastrointestinal anomalies,
munologic deficiency, and the sequence of acquisition
infant enteric flora. (*C. botulinum* does not appear to be
art of the normal
estinal flora.) The possibility of bacteriophages
nverting nontoxigenic *Clostridia* into toxigenic strains
s also been raised.[11] These studies need further
estigation.

The differential diagnosis of infant botulism includes
rious infections, toxins, and metabolic disorders of the
rvous system. However, as the disease progresses, a
turbance at the myoneural junction becomes more
parent, and the possibilities of organophosphate
isoning, black widow spider and snake toxin,
asthenia gravis, Eaton-Lambert syndrome, and
illain-Barre-Strohl syndrome are raised. (Figure 2).

The laboratory diagnosis of infant botulism depends
the demonstration of botulinal toxin and/or *C.*
ulinum organisms in fecal or serum specimens.
though detection of circulating botulinal toxin has
en frequently observed in the adult form of the
sease, serum-positive toxin has only been
monstrated in one infant to date (Case 3 of this
port). The organism type is verified by using specific
titoxins in the mouse toxin neutralization test. Since
culture of *C botulinum* from colon contents or blood
quires anaerobic procedures and special enrichment
chniques, proper collection of specimens is
cessary.[11] Information on laboratory facilities in the
ited States performing assays for botulinal toxin or
tures can be obtained from the Enteric Disease
vision, Center for Disease Control, Atlanta, Georgia
333.

Treatment of infant botulism is predominantly
pportive with particular attention to pulmonary and
tritional aspects. The need for vigorous pulmonary
treatments cannot be overemphasized. Although the prognosis in general is good, complete recovery may be prolonged (Case 3), and two deaths have been reported secondary to respiratory complications. The role of antibiotics is unclear. Theoretically, antimicrobials may be expected to inhibit futher intestinal colonization and, in turn, decrease the in vivo proudction of toxin. This, however, has not been supported by studies, and excretion of both the toxin and organism has been reported in two cases for as long as 138 and 158 days after a course of antibiotic therapy.[4] Controversy regarding the use of antitoxin in infant botulism also exists. Although the use of antitoxin is helpful in the adult form of the disese, no effect has been demonstrated in infants, and its use may cause the increased release of membrane-bound toxin and may even produce more severe symptomatology.[12] The use of Guanidine by its ACh release action may be of some value but merits further investigation and trial in infants.

Conclusion

In view of the universal distribution of *C. botulinum*, the need to be aware and recognize infant botulism cannot be overemphasized. The diagnosis should be entertained in any infant presenting with acute hypotonia and weakness. Diagnostic features have been reviewed. Although supportive and symptomatic care is the main mode of therapy at the present time, more specific treatment measures may be delineated in the future.

Acknowledgements

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Psychiatric Disorders

Cardiovascular Effects of Phenothiazines A Review

Mohammad A. Habibzadeh, M.D., FACP

Phenothiazines are among the most widely used drugs in the practice of medicine today. They are employed primarily in the treatment of psychiatric disorders. The agents are also used for their antiemetic and antihistaminic actions. However, the drugs have significant direct and indirect effects on the cardiovascular system. The untoward cardiovascular effects of phenothiazines are known to occur most frequently in patients with preexisting cardiac disorders,[1] following overdose ingestion of the drugs,[2] and in those receiving long term high dose therapy.[3, 4, 5] Phenothiazines have a wide range of therapeutic safety. However, although very rare, potentially hazardous cardiovascular complications have been reported following the administration of commonly employed doses.[6] The majority of the patients presenting with the untoward cardiovascular effects of phenothizines are managed by practicing physicians in emergency rooms and intensive care units. Therefore, familiarity with the cardiovascular effects of these compounds is the key to success in proper diagnosis and therapy of such patients.

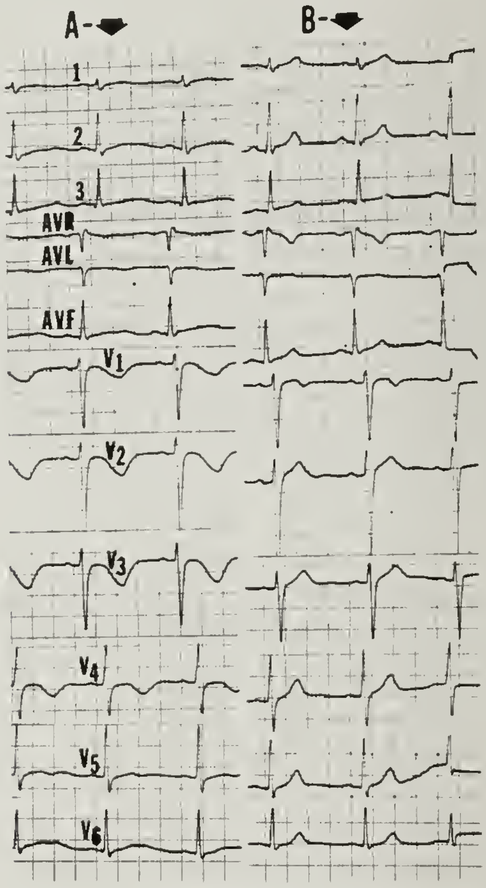

Figure 1
(A) Electrocardiogram of a 29 year-old male, obtained hours after an overdose ingestion of 3500 mg of mesoridaizi besylate, demonstrates flat T-waves in the infero-later leads, inverted T-waves in anterior precordial leads, and Q interval prolongation. (B) Electrocardiogram, obtained month later, is within normal limits.

Clinical Manifestatons:

Electrocardiographic (EKG) abnormalitie arrhythmias and hypotension are the most common si effects of phenothiazines.[7] The drugs, particularly t piperdine derivatives (thioridazine and mesoridazine), therapeutic doses cause EKG alterations (Figure including flattening or notching of the T-wav increased amplitude of the U-wave, T-wave inversio St-segment depression, and P-R, QRS, and QT-interv prolongations.[6, 8, 9] The most common EKG changes a alterations in the T-wave and ST-segment, occuring 50 to 70% of patients treated with these agents.[3] T changes are considered to be benign and reversible aft discontinuation of the drugs or administraton supplemental potassium salts.

There is much controversy regarding the occurren of sudden and unexplained death in patients receivin therapeutic doses of phenothiazines. However, clinic evidence strongly indicates that these agents, in tox doses, can cause serious cardiac arrhythmias such ventricular tachycardia and fibrillation, atrioventricular conduction disorders to the point heart block, with fatal termination.[4, 6] The incidence cardiovascular side effects is far more frequent individuals with preexisting cardiac disorders.[1] Becaus of this toxicity, there is relative contraindication to th use of phenothiazines in patients with cardiovascula disorders.

From: Veterans Administration Medical Center, Phoenix, AZ 85012. Dr. Habibzadeh, Chief, Cardiovascular Section and Director, Medical and Coronary Intensive Care Units.

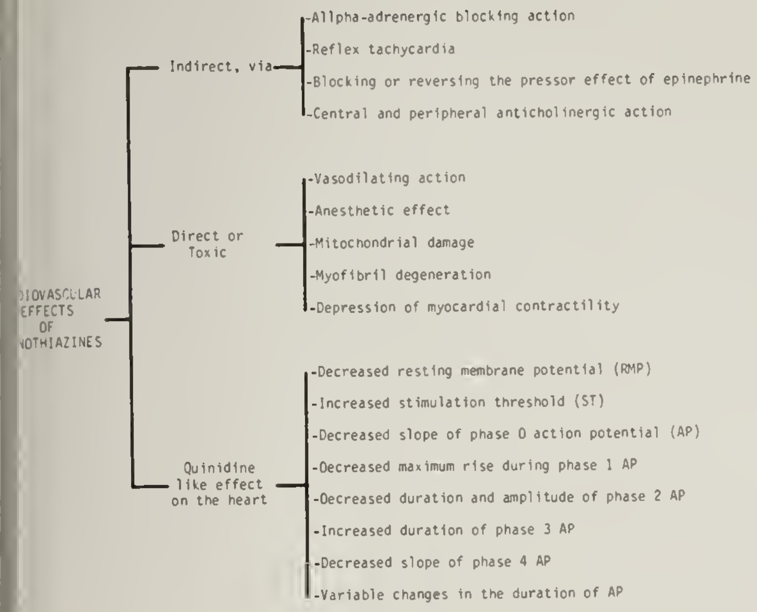
IOVASCULAR
EFFECTS
OF
IOTHIAZINES
Indirect, via
-Allpha-adrenergic blocking action
-Reflex tachycardia
-Blocking or reversing the pressor effect of epinephrine
-Central and peripheral anticholinergic action
Direct or
Toxic
-Vasodilating action
-Anesthetic effect
-Mitochondrial damage
-Myofibril degeneration
-Depression of myocardial contractility
Quinidine
like effect
on the heart
-Decreased resting membrane potential (RMP)
-Increased stimulation threshold (ST)
-Decreased slope of phase 0 action potential (AP)
-Decreased maximum rise during phase 1 AP
-Decreased duration and amplitude of phase 2 AP
-Increased duration of phase 3 AP
-Decreased slope of phase 4 AP
-Variable changes in the duration of AP

Figure 2
grammatic demonstration of various effects of nothiazines on the cardiovascular system.

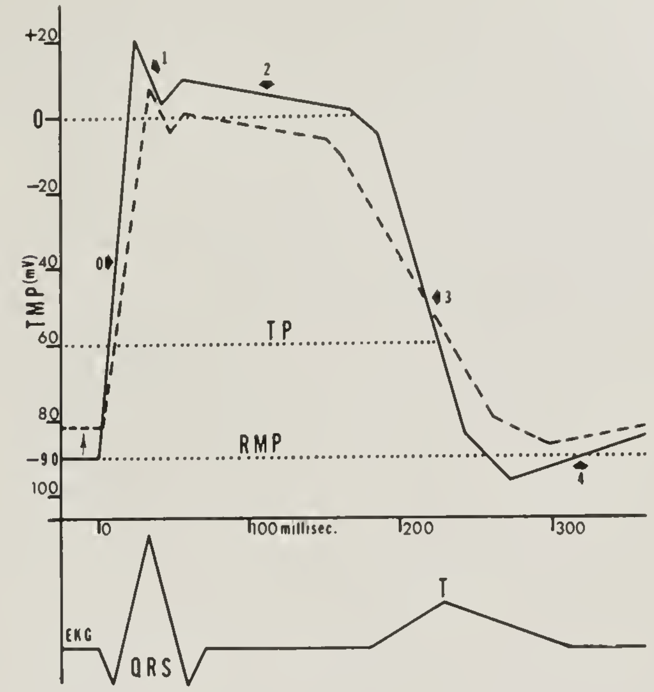

Figure 3
Schematic diagram, comparing the action potential (AP) of a normal cardiac purkinje cell (solid lines) with one demonstrating the effects of phenothiazines (broken lines). TMP, transmembrane potential; TP, threshold potenial; RMP, resting membrane potential.

chanism of Action:

he effects of phenothiazines on the cardiovascular tem are complex (Figure 2). They produce direct ect on the heart and blood vessels and indirect effect ough actions at all levels of central, peripheral and onomic nervous systems.[7] The drugs cause hostatic hypotension and syncope via a combination central and autonomic nervous system actions, ripheral alpha-adrenergic blockade, reflex hycardia, and a direct effect on the blood vessels. enothiazines have a so called quinidine like effect on heart, causing depression of conduction velocity and nporal dispersion of action potential duration in ious types of cardiac fibers[6, 10] (Figures 2 & 3). This reases nonhomogenous repolarization of different d closely adjacent cardiac fibers, which may edispose the heart to reentrant arrhythmias.[9] These npounds, due to potent central and peripheral ticholinergic effects, inhibit sino-atrial and ioventricular cholinergic stimulation, and allow compensated sympathetic nervous system effect on myocardium, which can cause sinus tachycardia and ner cardiac arrhythmias.[2] Phenothiazines shift the rve of membrane responsiveness to the right and crease dv/dt (amplitude of phase 0 action potential/its te of rise) for any given membrane potential[10] (Figure Unlike quinidine, these drugs prolong the effective fractory period in the ventricular muscle, while ortening it in purkinje fibers.[9] The drugs alter ycardial catecholamines and have direct depressant d cardiotoxic effects on the heart, which may cause rdiomegaly and congestive heart failure.[3, 5] However, eir effect on mycardial contractility is less than that oduced by therapeutic doses of quinidine.[9]

eatment:

Phenothiazines are highly bound to plasma proteins (90%) and are slowly metabolized by the liver. Since these agents, once absorbed, are not removed by the kidney or dialysis, the therapy in case of overdose is mostly symptomatic. This includes the control of cardiovascular and nervous system complications until the agents are metabolized and concentration is brought to nontoxic levels. Patients should have constant electrocardiographic monitoring until their condition is completely stable. Anticholinergic effects of phenothiazines on the central nervous system should be treated with physostigmine, which passes the blood brain barrier rapidly and reverses the phenothiazine induced central anticholinergic effects.[11, 12] Initially 2 mg of physostigmine is given by intramuscular injection or preferably by slow intravenous (IV) infusion. Maximum effect should appear within 20 minutes after the intramuscular and 10 minutes after the IV administration of the drug. Provided the drug proves effective, a 2 mg dose may be repeated every two hours if necessary. The use of physostigmine is relatively contraindicated in patients with coronary heart disease and conduction defects.[11] Excessive use of physostigmine causes cholinergic complications which should be treated with a parenteral quaternary ammonium anticholinergic agent such as glycopyrrolate (Robinul). Atropine and related compounds which penetrate the blood brain barrier easily, should be avoided, since they will potentiate the central anticholinergic effects of phenothiazine intoxication.[11, 12] The specific effect of physostigmine in phenothiazine induced cardiac arrhythmias is not known at this time. If cautious infusion of IV fluids is unsuccessful in correcting the phenothiazine induced hypotension, administration of an alpha-adrenergic agent like norepinephrine may be required. Epinephrine and other vasoactive agents with significant beta-adrenergic activity should be avoided, as they will potentiate the hypotension.[13]

Phenothiazine induced arrhythmias should not be treated with quinidine and procaine amide, for they may further reduce conduction velocity and enhance the arrhythmia.[6] Ventricular tachyarrhythmias should be treated with lidocaine and/or direct counter shock. If these measures fail to control the ventricular tachycardia, IV administration of propranolol hydrochloride, 0.1 mg/min may prove effective.[6] Under these circumstances, a transvenous temporary cardiac pacemaker should be inserted to control the heart rate in case significant bradycardia follows propranolol administration. Temporary cardiac pacing should be considered for therapy of symptomatic bradycardias and tachyarrhythmias unresponsive to conventional antiarrhythmic agents. This method can reduce the risk of further cardiac complications resulting from excessive administration of antiarrhythmic drugs in the presence of phenothiazine toxicity. Cardiac failure, occuring as the result of phenothiazine toxicity, should be treated with digitalis glycosides and diuretic agents.

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Radiology

Case of the Month No. 39

Jonathan M. Levy, M.D.
Edward I. Alper, M.D.
Cary J. Stegman, M.D.

Editor:
John C. Bjelland, M.D.

A 62 year old lady has complained of right uppe quadrant pain for several years. She has previously had cholecystectomy, but her symptoms have not improvec A radionuclide liver scan is markedly abnormal. C scans of the patient 's liver (Figure 1) and kidney (Figure 2) are shown.

What is the diagnosis?

What is the patient's prognosis?

Continued on Page 53

From: Scottsdale Memorial Hospital, Departments of Diagnosti Radiology and Gastroenterology, 7400 E. Osborn Rd., Scottsdale, AZ 85251.

State of the Art

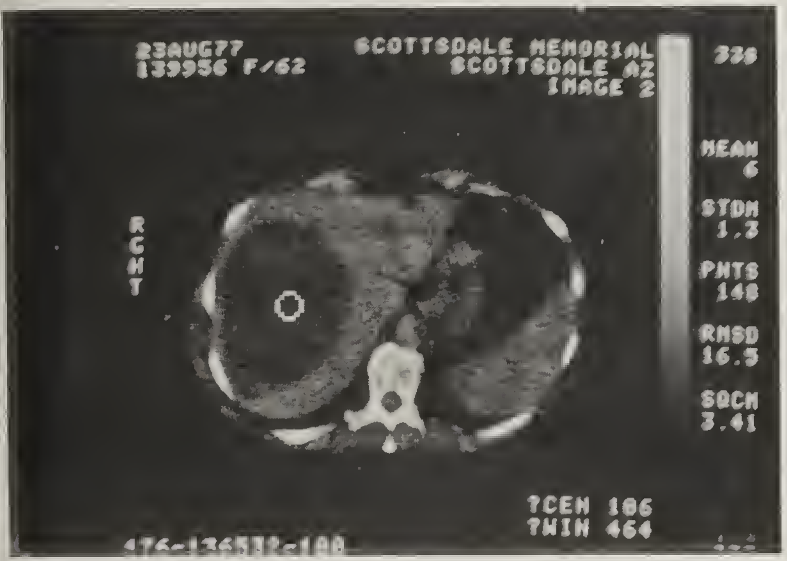


Figure 1
CT scan through liver. Note several areas of decreased density. Electronic cursor reads tissue density of largest lesion (MEAN = 6 in right upper corner).

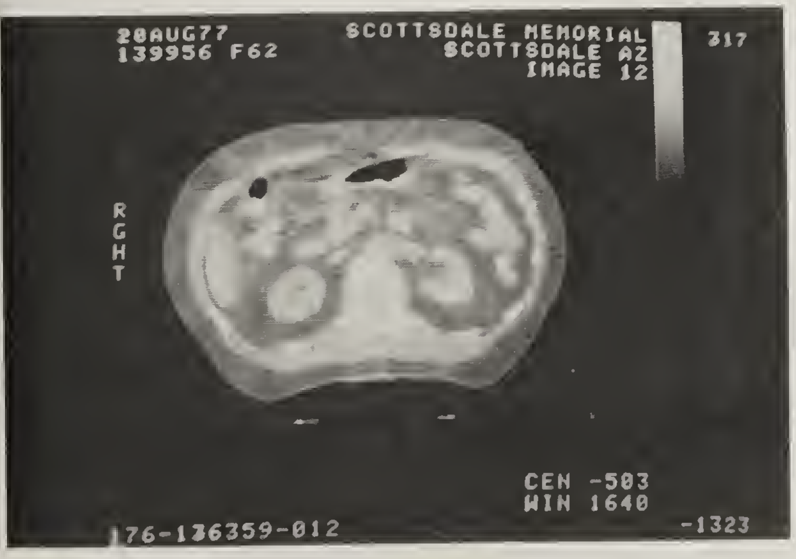


Figure 2
CT scan through kidneys.

Current Concepts in Bile Secretion and Cholestasis

David L. Earnest, M.D.

Eric H. Prosnitz, M.D., Editor

Introduction

Although jaundice resulting from failure of bile secretion has attracted the attention of physicians since at least the time of Hippocrates, only recently has much concerted effort been directed toward defining both normal mechanisms which control bile secretion and those which lead to impaired bile formation and cholestasis. For years, confusion in communication between clinician, pathologist and basic physiologist resulted largely from differing use of the term cholestasis. To the pathologist, cholestasis referred to histologic evidence of retained bile in canaliculi and ductules with bile pigment in liver cells. To the physiologist it connoted impairment of processes controlling bile formation and flow. To the clinician, cholestasis dentoed a syndrome accompanied mainly by jaundice and pruritis with abnormal tests of hepatic function. Although all of these perspectives are correct, it is more useful to define cholestasis simply as impairment of bile secretion with retention in blood and tissues of compounds normally excreted in bile. This definition does not specify the many and varied consequences of bile secretory failure.

From: University of Arizona Health Sciences Center, Dept. of Internal Medicine, Sect. of General Medicine, Tucson, AZ 85724.

Cholestasis could result from abnormalities at the molecular or cellular level or from obstruction to bile flow anywhere along the biliary tract. The studies of Eppinger in the 19030's led to the important realization that the biochemical and hepatic morphological chagnes which develop during complete extrahepatic obstruction are essentially the same as those seen with many liver diseases without any abnormality of the extrahepatic biliary system. Thus, an often difficult diagnostic problem has been the determination of whether jaundice and cholestasis is due to intrahepatic impairment of bile formation or extrahepatic obstruction to bile flow. In the past this difficulty was compounded by lack of precise clinical ways to evaluate patency of the extrahepatic biliary tree and from confusion about causative mechanisms and pathology of intrahepatic cholestasis. However, in the past 10 years, results of extensive research into mechanisms of bile formation and cholestasis and development of new diagnostic techniques have greatly increased our understanding of and ability to properly deal with cholestatic syndromes. The purpose of this article is to briefly review current concepts regarding mechanisms of bile formation and cholestasis. In addition, a convenient manner for classifying patients with cholestatic liver diease is discussed and methods currently useful in differentiating intrahepatic from extrahepatic cholestasis are enumerated.

Normal Bile Formation

Bile is a complex fluid containing principally water, electrolytes, bile salts, cholesterol, lecithin, bilirubin and a variety of metabolic products, ingested medications and dietary constituents excreted by the liver. Just as the glomerulus is the site of urine formation in the kidney, the canaliculus is the site of bile formation in the liver. Also, just as the renal tubules modify the glomerular filtrate, the biliary ductules modify canalicular bile. The functional unit of bile formation is called the hepatic acinus.[1] Blood from the hepatic arteriole and portal venule enters the acinar unit at the portal traid, courses through the sinusoidal space between liver cells and leaves via the hepatic venule. Compounds destined for excretion in bile are removed from sinusoidal blood by absorption or diffusion into parenchymal cells and transported into the biliary canaliculus. Canaliculi run parallel to sinusoids and join with and empty into cholangioles or intralobular bile ductules at the edge of the portal triad. Thus, bile flows in the canalicular space in a direction opposite to sinusoidal blood flow.

The canalicular space is formed between plasma membranes of two adjacent liver cells (Figure 1). The surface area is large and is incresed by small microvilli which project into the lumen. The extent of this space is limited by the cell plasma membranes and by specialized areas along the intracellular cleft known as tight junctions. These junctions form barriers between the canalicular space and the intercellular cleft which is continuous with the sinusoidal space. The canalicular space is thus distinctly separate form the sinusoidal space. Water and other molecules destined to appear in bile must traverse either the liver cell or pass through the intracellular junctional complex. The important studies of Brauer which utilized an isolated perfused liver system clearly showed that in contrast to urine formation by the kidney, hepatic bile formation does not result from hydrostatic pressure.[2] These observations led to the suggestion that water movement for bile formation must result from osmotic gradients generated by active transport of solute into the canalicular space.[3] A considerable number of investigations have led to the current concept that active transport of bile salts into the canaliculus is the major driving force for bile formation.[4] Bile resulting from active bile salt transport is known as bile salt dependent bile flow.

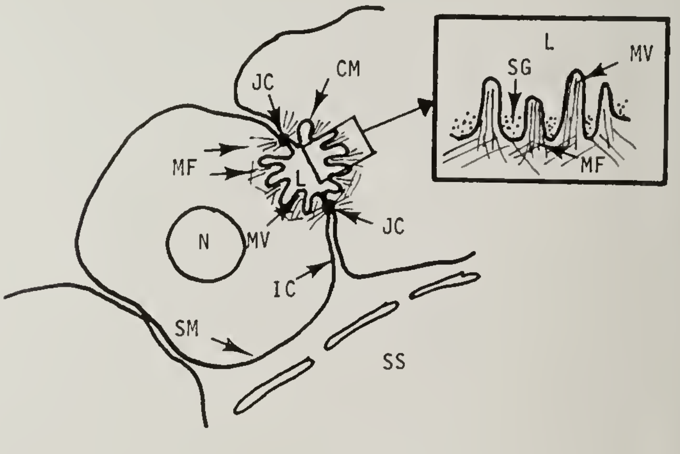
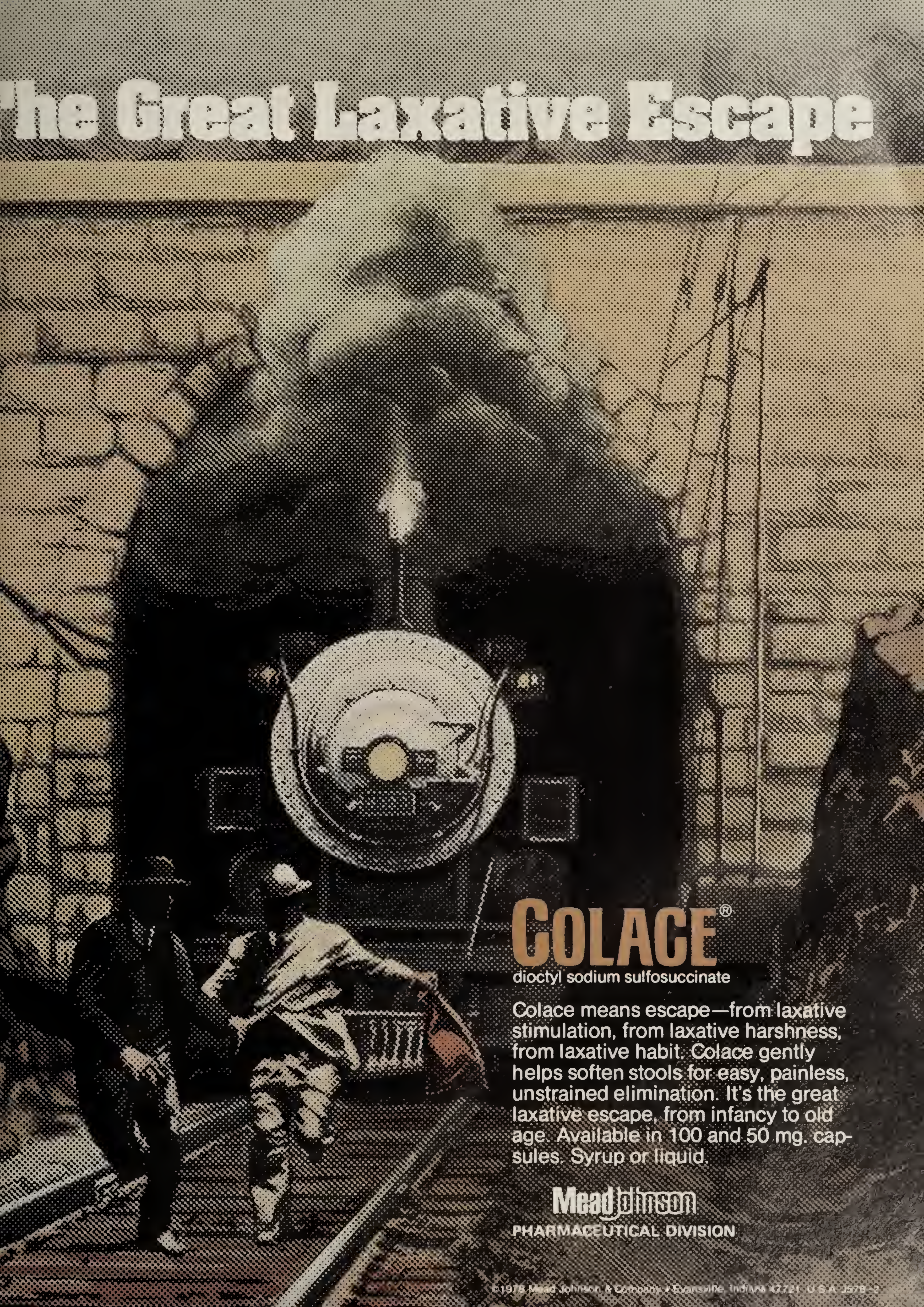


Figure 1
Schematic drawing of canaliculus and liver cell. Expanded area of canalicular membrane (CM) shows microfilaments (MF) supporting the microvillus (MV). It has been postulated that active ion transport creates a standing osmotic gradient (SG) at the base of the microvilli. N-nucleus, JC-junctional complex, IC-intercellular cleft, SM-sinusoidal membrane of liver cell; SS-sinusoidal space, L-canalicular lumen.

There is an excellent linear correlation between bile flow and bile salt excretion in all species studied. The physiologic basis for this linearity is the osmotic properties of the bile salt micelle.[5] It is important to understand that osmotic force is exerted by the micelle as a unit and not by the component bile salt molecules. For example, exretion of bile salts which do not form micelles, such as dehydrocholate, produces a much greater volume of biliary water flow or choluresis than that which occurs with excretion of normal human bile salts. Although dehydrocholate stimulates formation of a large bile volume, excretion of cholesterol and phospholipid is not increased. Thus, with excretion of normal micelle-forming bile salts, bile salt dependent bile flow is proportional to both bile salt excretion rate



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COMPATIBILITY

Eastern Tiger Swallowtail Butterfly
(Papilio glaucus)

Does it influence your choice of a peripheral/cerebral vasodilator*?

Vasodilan–compatible with coexisting diseases (e.g., glaucoma, diabetes)

Vasodilan has not been reported to affect the course of coexisting disease; it has not been reported to affect blood sugar levels or to raise intraocular pressure.

Vasodilan–compatible with concomitant therapy

Vasodilan has not been reported to affect the treatment of coexisting disease; it is compatible with such drugs as hypoglycemics and miotics.

Vasodilan–compatible with your total regimen for vascular insufficiency

Vasodilan can be a valuable adjunct in planning a total therapeutic program for vascular insufficiency.

***Indications:** Based on a review of this drug by the National Academy of Sciences-National Research Council and/or other information, the FDA has classified the indications as follows:

Possibly Effective:

1. For the relief of symptoms associated with cerebral vascular insufficiency.
2. In peripheral vascular disease of arteriosclerosis obliterans, thromboangiitis obliterans (Buerger's Disease) and Raynaud's disease.

Final classification of the less-than-effective indications requires further investigation.

Composition: Vasodilan tablets, isoxsuprine HCl, 10 mg. and 20 mg. Vasodilan injection, isoxsuprine HCl, 5 mg., per ml.

Dosage and Administration: Oral: 10 to 20 mg., three or four times daily. Intramuscular: 5 to 10 mg. (1 or 2 ml.) two or three times daily. Intramuscular administration may be used initially in severe or acute conditions.

Contraindications and Cautions: There are no known contraindications to oral use when administered in recommended doses. Should not be given immediately postpartum or in the presence of arterial bleeding.

Parenteral administration is not recommended in the presence of hypotension or tachycardia.

Intravenous administration should not be given because of increased likelihood of side effects.

Adverse Reactions: On rare occasions oral administration of the drug has been associated in time with the occurrence of hypotension, tachycardia, nausea, vomiting, dizziness, abdominal distress, and severe rash. If rash appears the drug should be discontinued.

Although available evidence suggests a temporal association of these reactions with isoxsuprine, a causal relationship can be neither confirmed nor refuted.

Administration of single dose of 10 mg. intramuscularly may result in hypotension and tachycardia. These symptoms are more pronounced in higher doses. For these reasons single intramuscular doses exceeding 10 mg. are not recommended. Repeated administration of 5 to 10 mg. intramuscularly at suitable intervals may be employed.

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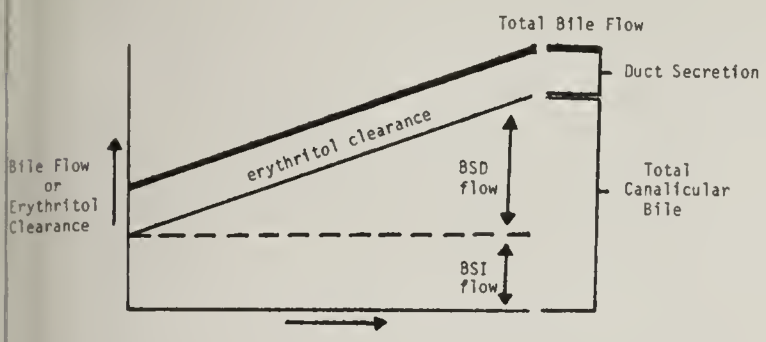

Figure 2
chematic drawing of relationship between bile fow or rythritol clearance and bile salt excretion rate. Extrapolation f erythritol clearance regression line to theoretical zero bile lt excretion defines magnitude of bile salt independent (BSI) ow. Bile salt dependent (BSD) flow makes up the difference total canalicular bile flow.

nd the number of micellar units formed.

Canalicular excretion of cholesterol is partly ependent on bile salt secretion. Excretion of hospholipid appears to be completely dependent on bile alt secretion. The exact mechanism responsible for ransport of bile salts into the canalicular space and with hem the transport or movement of cholesterol and hospholipid is unknown. It is possible that these species ither cross the canalicular membrane as part of repackaged micelles or move as individual monomers vith aggregation in the canalicular lumen. lternatively, biliary cholesterol and phospholipid could e derived from canalicular membrane structure and be emoved during bile salt transport.

Bile salts are actively transported into the canaliculus nd specific bile acid binding sites have been identified in he canalicular membrane.[6] In addition to that for bile cids, there are at least three additional separate active arrier mechanisms which transport organic molecules nto bile.[7] One is for other organic anions such as BSP, ilirubin and iopanoic acid; one is for organic cations uch as procainamide; and one is for neutral compounds. Bile flow and the secretory rate (or transport maximum) nto bile of various anionic compounds can be greatly enhanced by increased bile acid secretion. However, increased canalicular transport of other organic anions uch as bilirubin does not itself significantly increase bile low. The explanation for this may be that such organic nions are probably transported in bile with bile salt nicelles and thus do not exert individual osmotic forces.

In summary, active transport of bile salts into the analiculus is the major driving force for canalicular vater flow and possibly also for excretion of bilirubin nd bile lipids. It seems reasonable to anticipate that any mpairment of bile salt transport will reduce bile flow nd excretion of other organic molecules and thus may e a central abnormality in the pathogenesis of holestasis.

In order to precisely measure canalicular bile formation and to differentiate it from bile which is added by bile duct epithelium, it was necessary to find a molecule which enters the canaliculus with hepatic cell water, which is not metabolized and which is not actively transported by the canalicular or bile ductular membranes. Mannitol and especially erythritol have been experimentally found to most closely fit these requirements. Accordingly, hepatic clearance of these solutes has been interpreted to reflect total water movement into the canaliculus and to be equivalent to canalicular bile formation.[8] The volume of canalicular bile is less than the total amount of bile which flows from the common bile duct. Additional bile volume results from water and electrolytes secreted by the biliary ductular epithelium. The ductular component of total bile flow is modified largely by various gastrointestinal hormones, especially secretin which increases water and electrolyte content of bile.[9]

Evaluation of erythritol clearance has demonstrated that canalicular bile flow consists of at least two components. One is directly related to bile salt secretion and is called the bile salt dependent (BSD) fraction. When erythritol clearance is plotted as a function of bile salt excretion and the obtained regression line extrapolated to zero, the intercept on the bile flow axis is positive. This suggests that at zero bile salt excretion, a volume of bile water is formed which is independent of bile salt secretion. The second component of canalicular bile formation is known as bile salt independent (BSI) bile secretion. It has been measured in man by a number of investigators and found to contribute approximatley 35% of the average total bile flow.[10]

The exact mechanisms responsible for BSI bile formation continue under intensive investigation. Canalicular and pericanalicular ion pumps, especially Na,K-ATPase are thought to play a major role in its formation. Initial histochemical studies suggested that Na,K-ATPase was located in the canalicular membrane itself.[12] Accordingly, it was postulated that active Na^+ transport into the canaliculus occurred directly across the canalicular membrane. However, more recent studies using refined staining methods have failed to show Na,K-ATPase primarily in the canalicular membrane but instead present mainly in the liver cell sinusoidal membrane, especially in the intracellular cleft above the junctional complexes.[13] This new observation suggests that transported Na^+ and osmotically obligated water which lead to BSI bile flow may enter the canalicular space by passage through the tight junctions. Although the exact route for Na^+ entry into the canaliculus is not certain, the fact that oubaine, which inhabits Na,K-ATPase activity, reduces BSI bile flow makes it very likely that active Na^+ transport is partly responsible for BSI flow.

Phenobarbital has been shown to increase BSI bile flow. The mechanism of its effect is uncertain but may result from enhanced synthesis of canalicular membrane components secondary to a general stimulatory effect of phenobarbital on liver cell protein synthesis. Increasing the hepatic concentration of cyclic AMP also increases BSI bile formation. The mechanism for this effect is believed to operate through enhanced Na^+ transport. Drugs such as hydrocortisone and theophylline and hormones like glucagon and secretin also increase BSI bile formation, possibly through their effect in increasing cyclic AMP. A component of BSI bile flow has also recently been shown to result from canalicular bicarbonate secretion.[14]

Finally, there is evidence which suggests that the cytoskeleton of the liver cell, especially that adjacent to the canaliculus, plays an important role in bile formation. Electron microscopic studies have shown 50-/0 A actin-containing filaments in the pericanalicular cytoplasm which attach to the lateral cell membrane at the area of the junctional complex and extend into the core of the canalicular microvilli appearing to give them structural support.[15] Erlinger has recently postulated that active solute transport into the canaliculus may occur specifically at the base of each canalicular microvillus and cause formation of a standing osmotic gradient.[16] This would cause bulk water flow into the canalicular lumen by a mechanism similar to that proposed for other secreting epithelium.[17] Since microfilaments appear to give support to the anatomic configuration of the canalicular membrane, distruption of the microfilament system might cause shortening or flattening of the microvilli and impair bile formation by compromising the standing osmotic gradient. Indeed, a number of compounds discussed below directly affect the microfilament system and cause cholestasis.

Mechanisms Causing Cholestasis

Partial or complete obstruction of the extrahepatic bile ducts obviously impairs bile flow and results in cholestasis. At the canalicular level, such obstruciton causes blunting or even absence of microvilli with marked widening of the canalicular space. It is not certain whether these changes result from increased intraductal pressure or develop secondary to membrane, cytoskeletal, etc. damage owing to other phenomena which accompany impaired biliary excretion. Since these same gross canalicular changes also are seen with cholestasis due to primary liver cell dysfunction, it seems likely that at least part of the canalicular abnormality is initiated by biochemical alterations in cellular function at the molecular level. However, it is not yet possible to specify which ractor or factors at this level lead to impaired bile formation. Table 1 lists potential causative mechanisms which have been suggested to be important. (Table 1)

Table I
Potential Mechanisms Causing Canalicular Cholestasis

1. Abnormal bile acid metabolism and transport.
2. Defect in other organic anion transport.
3. Interference with mitochondrial energy supply.
4. Impairment of Na+ ion transport.
5. Abnormal canalicular membrane permeability.
6. Impaired microtubule and microfilament function.
7. Abnormal permeability of hepatic junctional complexes.

It has frequently been hypothesized that an abnormality in canalicular bile salt transport plays a central role in causing cholestasis. However, no primary or specific defect in bile salt transport per se has yet been defined. In an experimental situation, infusion of excessive amounts of certain monohydroxy bile salts such as lithocholate, 3-hydroxy-5-cholenoate, and also the dihydroxy bile salt chenodeoxycholate leads t impaired canalicular transport of other bile acids, cause distinctive abnormalities in the canalicular membran and produces cholestasis.[4, 5] Canalicular membran abnormalities attributed to excessive amounts o cholestatic bile salts include deposition of the bile salt themselves in the membrane, increased membran cholesterol content and a change in membrane fluidit and configuration.[18] Thus, certain bile salts may produc cholestasis by causing canalicular membran dysfunction. Whether or not there is increased hepati formation of monohydroxy bile acids in cholestatic live disease as has been postulated is unknown but availabl data suggest this is unlikely.[19, 20]

Another mechanism which could play a role in causin cholestasis in liver disease is impaired formation of bil salt sulfates. In cholestasis the liver forms sulfate ester of bile salts, a process which makes them more wate soluble and enhances their excretion in stool and urine.[2 It is possible that with certain types of hepati dysfunction, there is impaired activity o cholylsulfotransferase, the enzyme responsible for bil salt sulfation.[22] This could result in hepati accumulation of cholestatic bile salts and thereby creat a vicious circle.

Defects in the transport of other organic anions als have been postulated as a cause for cholestasis bu convincing evidence for this is lacking. For example, i the Dubin-Johnson syndrome there is defectiv canalicular transport of conjugated bilirubin. However bile flow is normal and despite the presence of bilirubi retention and jaundice, true cholestasis is absent Alterations in mitochondrial structure and functio have also been observed in cholestasis.[23] Most cellula ATP, a source of metabolic energy, is synthesized i mitochondria. Reduced synthesis of ATP as a result o liver cell damage could favor development of cholestasi since hepatic uptake, metabolism and scretion of bil acids all require metabolic energy.[24]

Impaired Na^+ transport into the canaliculus also coul reduce bile formation. Certain cholestatic drugs such a ethinyl estradiol cause a decrease in liver cell membran Na,K-ATPase activity.[25] It is not clear whether thi change is due to reduced membrane enzyme content o to a change in enzyme activity of the protein. Obviousl any factor which affects integrity of canalicula membrane structure could affect its enzyme activity an transport function. For example, increase in canalicula embrane total cholesterol content occurs with urolithocholate induced cholestasis and membrane ntent of unesterified cholesterol is increased with hinyl estradiol cholestasis.[26] Lithocholate and hlorpromazine both appear to deposit in the canalicular embrane and modify its structure. Both cause holestasis.

The potential key role of the cytoskeleton in cilitating bile formation has been mentioned. In rats, e fungal alkaloid cytochalasin B causes B/dose-related sruption of pericanalicular microfilaments, dilatation the canaliculus, loss of microvilli and cholestasis.[27] hese changes are all similar to those characteristic of trahepatic cholestasis in man. It is thought that tochalasin B causes disruption of microfilaments by ocking the association of cytoplasmic actin with actin-nding protein in the liver cell plasma membrane. halloidin, one of the toxic principals in the mushroom *manita phalloides,* also produces severe cholestasis ffecting a change in microfilaments. Even small mounts of this compound produce a striking ccumulation of microfilaments in the pericanalicular pace which cause distortion of the canalicular nembrane. Thus, it is possible that factors which rimarily affect production and stability of the actin-ontaining microfilament cytoskeleton may modify bile ormation.

Cholestasis could functionally result both from npaired secretion of bile into the canalicular space as vell as from abnormal return of bile constituents from analiculus to sinusoidal blood. Very interesting recent tudies have shown that bile acids themselves can affect lux through the junctional complex.[29] It is possible that primary abnormality in the tight junction may lead to iochemical evidence of cholestasis without actual mpairment in canalicular membrane transport.

Many or all of the above-mentioned abnormalities lay a contributing role to impaired bile formation in holestatic liver disease. Which, if any, if of greatest mportance or is the initiating factor unknown. It is onceivable that there are many routes to a final ommon pathway which is cholestasis and that each of he above mechanisms could serve as an entry.

Consequences of Cholestasis

Cholestasis is characterized by failure to excrete a number of substances normally secreted in bile. Retention of these compounds produces almost predictable consequences which in general are not diagnostically specific for an individual cholestatic disease. For example, impaired transport of organic anions causes 1) elevation of serum and tissue concentrations of bile acids thought to be somehow responsible for pruritis, 2) bilirubin retention leading to jaundice and, 3) faliure to excrete iodinated biliary x-ray contrast agents. Impaired hepatic bile acid excretion also leads to diminished concentration of bile salts in the intestinal lumen which in turn results in malabsorption of dietary fat and fat soluble vitamins. Malnutrition and the consequences of vitamin A, D and K deficiency may become clinically important in those cholestatic diseases where protracted cholestasis is to be expected, for example in primary biliary cirrhosis and chronic hepatitis. In such situations, cholestasis itself may be the pathogenesis of significant clinical problems.

Clinical Diagnosis of Cholestasis

Cholestasis is usually first recognized clinically by bilirubin retention, jaundice and pruritis. However, the most sensitive reflection of impaired bile secretion is elevation of bile acid concentration in serum. Serum bile acid concentration can be abnormal long before bilirubin retention is clinically evident. The liver normally extracts almost all bile acids from portal blood resulting in very low concentration of bile acids in the peripheral circulation. Due to active movement of the bile acid pool through the enterohepatic circulation with meals, the post-prandial concentration of bile acids in peripheral blood is slightly increased compared to that during fasting. This phenomenon is magnified in patients who have even minimal degrees of hepatic cell dysfunction. Such persons have markedly increased post-prandial concentrations of serum bile acids. Test kits are commercially available which allow easy measurement of serum bile acid concentration by utilizing radioimmunoassay. It has been suggested that the level of serum bile acids may be clinically helpful in diagnosis and management of certain types of liver disease, for example chronic hepatitis. It would appear not to be of great value when hepatic dysfunction has progressed to clinical jaundice since cholestasis is then obviously present. More experience is needed with serum bile acid levels in specific disease entities before their true value in clinical practice can be accurately assessed.

Cholestais is also associated with increased synthesis and release of enzymes from the liver plasma membrane. Increases in the hepatic fraction of alkaline phosphatase and of 5′-nucleotidase have been considered helpful in reflecting presence of cholestasis. More recently, another enzyme, gamma glutamyl transpeptidase (GGT), which is present in a number of body tissues, has been found to be elevated primarily in liver disease and to be remarkably high in obstructive liver disease.[30] Unfortunately, as with other enzymes which reflect cholestasis, it will not accurately differentiate between intrahepatic and extrahepatic obstruction.

Clinical Classification and Differential Diagnosis of Cholestatic Liver Disease

The majority of causes for cholestasis are from intrahepatic diseases. The classification for cholestatic liver disease shown in Table II is modified from that suggested by Popper and Schaffner.[19] (Table II) Obstruction of biliary radicals inside the liver may produce a picture indentical to that of extrahepatic obstruction. However, it should be remembered that only about half of the normal liver mass is required to

Table II
Clinical Causes of Cholestasis[19]

A. Extrahepatic Biliary Obstruction
   1. Ampulla of Vater
   2. Pancreatic Head
   3. Common bile duct (tumors, sclerosis, Lymph nodes)
   4. Porta hepatitis

B. Intrahepatic Biliary Obstruciton
   1. Focal abscess, carcinoma, cyst, parasites
   2. Intrahepatic biliary atresia
   3. Suppurative and non-suppurative cholangitis
      a. Bacterial
      b. ?immunologic
         —primary biliary cirrhosis
         —pericholangitis with inflammatory bowel disease.

C. Intrahepatic Cholestasis with Morphologic Changes of "Hepatitis"
   1. Viral
   2. Alcoholic
   3. Drug
   4. Cholestasis in Infancy - neonatal hepatitis
   5. Sepsis

D. Intrahepatic Cholestasis without Morphologic Changes of "Hepatitis"
   1. Benign recurrent intrahepatic cholestasis (Summerskill-Walsh Syndrome)
   2. Byler's Disease
   3. Recurrent jaundice of pregnancy
   4. Drug induced cholestasis
   5. Post operative cholestasis
   6. Cholestasis with lobar pneumonia & passive hepatic congestion
   7. ? mild forms of viral hepatitis

E. Diseases with elevated Direct Reacting Bilirubin without Cholestasis
   1. Dubin Johnson Syndrome (black pigment in liver)
   2. Rotor Syndrome (no black pigment in liver)

excrete the daily bilirubin load. Because of this, considerable intra or extrahepatic bile duct obstruction may be present and serum bilirubin remain normal. In this situation alkaline phosphatase, 5′-nucleotidase and GGT are usually quite increased. Cholestasis associated with hepatitis-type of histologic reaction can obviously occur from a wide variety of causes. In older individuals, a drug-related etiology should receive high consideration.

It should be remembered that persons may have increased serum bilirubin without liver disease or cholestasis. Increase in only the indirect reacting fraction should raise the possibility of hemolysis or Gilbert's syndrome. A persistent elevation of direct reacting bilirubin most commonly reflects liver disease but may be due to the Dubin-Johnson syndrome.

Correct diagnosis of the cause of cholestasis requires careful general medical assessment and usually serial evaluaiton of tests reflecting hepatic functions. If evidence of cholestasis persists or worsens, there are a number of ways to proceed. Extrahepatic biliary obstruction can now be easily evaluated, but not always excluded by use of ultrasound, computed tomographic scans and even newer radionuclide procedures which quantitate biliary excretion of technesium-labelled compounds.[31] If necessary, more precise evaluation for intra or extrahepatic ductal disese can be made endoscopic retrograde cholangiopancreatograph (ERCP) or by percutaneous transhepat cholangiography (PTC). ERCP has the advantage allowing concomitant visualization of the pancrea ducts and direct inspection of the ampulla of Vate However, in addition to cost, time and comfo advantages, PTC has the distinct advantage visualizing the biliary tree above the level of duct obstruciton. This is often important in plannin corrective surgery.

The cause of intrahepatic cholestasis is best evaluate by examination of liver tissue. Recent studies hav reaffirmed the safety of percutaneous needle biopsy the liver in the absence of contraindicating factor mainly coagulation abnormalities.[32] Liver biopsy unde laparoscopic control has been in common use in Europ for more than a decade. Its application in this country increasing, especially where focal hepatic lesions ar suspected. A more detailed discussion of specifi cholestatic diseases of the liver and of the best diagnosti approach for each is beyond the scope of this paper.

Summary

For years, cholestasis has presented major difficultie in clinical differential diagnosis. In the past few years much has been learned about normal physiology which controls bile formation and about factors which impai these mechanisms. Recongition that consequences o cholestasis produce a syndrome rather than manifestations of a specific disease has allowed a more rational basis for pursuing differential diagnosis Remarkable technologic advances have helped with earlier diagnosis of obstructive liver disease and to allay concern for unrecognized extrahepatic obstruction as the cause for persisting cholestasis. However, as is often the case, today's state of the art in cholestatic liver disease has raised as many questions and new possibilities as it has answers. These should assure continued progress in the field.

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Balance of references available upon request.

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Polycystic Liver Disease

Multiple low density lesions are seen in liver (Figure 1). These are smooth walled, and have a tissue density equivalent to that of water (+ 10 Units on our CT scanner). There is no evidence of dilatation of the hepatic ducts. These findings indicate the diagnosis of polycystic liver disease.

Polycystic liver disease is relatively uncommon condition, occurring in one out of 351 to 773 autopsies. Approximately 59% of the patients with hepatic cysts will have polycystic renal disease. (Our patient does not-see Figure 2) a small percentage of patients will also have cysts in the pancreas, spleen and/or lungs. Additionally, there is a strong association between polycystic disease and central nervous system aneurysms. Cystic liver diease is four times as common in women as in men, in contrast to an equal sex distribution in cystic kidney disease. Liver function is almost invariably normal. Most patients are asymptomatic, the cysts being found incidentally or after the discovery of renal cysts. Those patients with symptoms may complain of pressure and distension in the upper abdomen, and less frequently of pain resembling gallstone colic.

Diagnosis of polycystic liver disease is best made by ultrasound or CT scan, since these modalities show the multiple lesions to be filled with fluid. Radionuclide liver scans will show multiple cold areas in the liver, but the scan is not specific for polycystic disease. Plain film tomograms of the liver after administration of IV contrast media may also show the cysts.

The prognosis for isolated polycystic liver disease is excellent, in contrast to the poor prognosis for polycystic kidney disease. Rare complications include rupture of cysts, hemorrhage, portal hypertension with varices, jaundice and malignant degeneration. Large, symptomatic cysts may be drained for relief of symptoms. Our patient had CT guided percutaneous drainage of the large hepatic cysts, which relieved her right upper quadrant pain.

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Auxiliary Highlights

The Immunization Bill, endorsed by the ArMA Auxiliary, passed and was signed into law by Governor Bruce Babbitt.

Under the new law, all children attending public school from pre-school through grade 12 must meet immunization requirements within the first two months of school this fall. The previous law, passed in 1976, required immunizations only for children entering kindergarten through eighth grade for the first time and allowed one year for compliance.

School districts will be asking parents for a record of each child's immunizations. The Pima Council to Immunize Arizona's Children, which sponsored the legislation, has printed enough Immunization and Health Record booklets so there should be one available for each school child in the state of Arizona. These booklets will be very handy as they are designed specifically for Arizona.

Members of the Medical Auxiliary are delivering 60,000 of the booklets and immunization brochures to doctors who give immunizations — to pediatricians, family doctors and GPs. We will provide them for any doctor who requests them. The booklets are also being distributed through hospitals and county health clinics.

If you need booklets for your office, call the Auxiliary contact in your county:

Coconino
Margo Kennerly, Flagstaff, 526-1342
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Jean Dew, Tucson 326-5453
Mary Habra, Tucson 298-7057

Walk-in immunization clinics will be held in August and September in most counties, to get in compliance with the new law. We urge all doctors who treat school age children to keep the requirements of the new law in mind as they see patients this summer and give school or camp exams. If you review the child's immunizations at that time, give any necessary immunizations, and record all immunizations in one of the booklets, it will help avert a log-jam come fall and school registration.

Mrs. Philip E. Dew
Mrs. Farouk Habra
ArMA Auxiliary
Immunization Committee

Teaming with Business n a Common Cause

Last November an important merican said in a speech delporing ederal over-regulation and "Big rother government":

"Will our people be tempted by 'quick ures' to major problems, through overnment action on symptoms .....?

The speaker continued, "A majority of mericans believe the cost of regulation utweighs its benefits. A majority also elieve government has become too aternal in trying to protect people from heir own actions or inactions ....

"Our challenge then is to recognize this ew public mood as an opportunity to ebuild American faith in the free market rinciples that are the source of the trength and durability of the American ay of life."

Who made these remarks to the Rotary lub of Chicago? A medical spokesman? ne well could have, in view of such citons as the Federal Trade Commission ttacks on physician-advertising rinciples and medical-school ccreditation procdures. And in view of he clouded legislation that becomes tormy regulation—as in the case of the ealth Planning Act of 1974.

But no, the speaker was William B. ohnson, chairman and chief executive fficer of a large conglomerate, IC ndustries, Inc.

What he said reflects the identity of nterest—including public interest—etween medicine and business.

A startling example of government elf-interest versus medicine, industry specifically, the pharmaceutical), and the ublic was HEW's January firing of Norman Latker as its chief patent counsel after 22 years of federal service.

Latker had testified to Congress that HEW was delaying the release of potentially life-saving drugs. The *Chicago Tribune* quoted him, "The worst thing I could have done as HEW might see it was to tell the truth ....

The AMA also has deplored the delays in the approval and distribution of potentially beneficial drugs.

Drug regulation is one of the key issues faced by the new Congress. Another that affects medicine, industry, and the public is health-care cost containment—with the threat of federal controls that could ultimately result in rationing of care.

Cost containment is an area in which medicine and industry have been cooperating and must futher cooperate. Industry was represented on the AMA-sponsored National Commission on the Cost of Medical Care, which (in the spirit of Mr. Johnson's "free market principles") stressed marketplace choice in health coverage. Industry also has an important role in the Voluntary Effort to curb the cost rise. And, AMA representatives have been talking to corporate boards of trustees on the cost problem and the profession's approaches.

Doctors in each state and locality should tell business (and the public) what our federation is doing about costs and other concerns—and invite joint voluntary action as opposed to federal hyperactivity.

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Walter V. Edwards, M.D.
C. Herbert Fredell, M.D.
Warren Gorman, M.D.
John R. Green, M.D.
John W. Kennedy, M.D.
William B. McGrath, M.D.
Stanley H. Nordomo, M.D.
Murray Spotnitz, M.D.
John Stoner III, M.D.
William C. Weese, M.D.
Donald J. Ziehm, M.D.

Correspondence

Editor
Arizona Medicine

Dear Dr. Block:

I have just finished a Mini-Residency in Anesthesiology offered by the University of Arizona's School of Medicine in Tucson. Thanks to the talents of Anthony F. Vuturo, M.D., Associate Dean for the Office of Continuing Medical Education and Outreach, and to Christy Snow, Coordinator of the Mini-Residency Program, Office of Continuing Medical Education, my experience was most exciting.

When it comes to accumulating Category I credits, I would say there is no better way to go. The Mini-Residency provides an opportunity for those of us in the rest of the State to get acquainted with the Medical School, its faculty, its students and its resources.

Outstanding features of the program are that a great freedom of choice is given to the Mini-Resident in putting his program together. There was also the utmost cooperation on the part of the department of Anesthesiology in assisting me to accomplish the objectives I had established for myself. In particular, I learned a method of using very expensive anesthetic agenst (Enflurane and Halothane) in a closed circuit. At today's costs this is extremely practical information, since it means that when put into effect, it will result in appreciable savings.

Apart from the extremely warm and helpful reception on the part of all members of the teaching and resident staff in anesthesiology, I had the opportunity to acquaint myself with the resources of a truly magnificent library. I only wish that such audio-visual aids as are available to today's students could have been available to my generation. The medline of course is something that all of us can use in our practices today. However until I visited the University, I was not aware of the many ramifications of this extremely useful feature of our University's library.

I noted with pleasure that in the classes I attended (and I sat in with students during the regular lectures in Physiology) that the basic science teaching was intended and designed to be clinically relevant.

This was such an extremely gratifying experience to me from every standpoint that I would like to bring the possibility of taking advantage of the Mini-Residency program to my Colleagues.

Wallace A. Reed, M.D.
Medical Director
Surgicenter

Errata

In the article *Percutaneous Transluminal Coronary Angioplasty: Can Cardiac Catherterization be Therapeutic?*, which appeared in the May issue of *Arizona Medicine* Vol 36, pages 353-355, 1979, the figures 2 and 3 are in reverse order. The legends remain correct. Our apologies to the author Lawrence P. Temkin, M.D.

In Memoriam

R.Lee Foster, M.D.

1907-1979

Like many others, Lee came to Arizo for health reasons—arrived in 1926. H health had failed, tuberculosis, while l was attending college in his native stat Illinois. After eighteen months in sanatorium, he came West for th "climate cure".

Fortunately his quest for health wa successful. He worked variously as a rad repairman, the manager of an electric supply house, he did a stint with radi station KFAD, now KPNX, attended th Gradwohl Medical Technology School i St. Louis. Then he moved to Los Angele to complete his premedical studies an graduated from USC Medical School i 1941. He returned to intern at St. Joseph Hospital, Phoenix—and then did preceptorship in radiology under W Warner Watkins, M.D. at th Pathological Laboratory and becam certified by the American Board c Radiology.

Lee came into radiology near the clos of an era. In many communities th pathology and radiology hospita departments were directed by the sam physician. Prior to WW II thes specialities became separate i postgraduate training and practice. Wit the many subspeciality divisions i pathology and radiology at the presen time, a super specialist in either of thes fields of restricted proficiency ca scarcely imagine a "general" radiologist o pathologist and for one physician t function in both fields simultaneously—ah well, so it once was.

Lee spent his professional career a Director of the Pathologic Laborator and its successor the Medical Cente Laboratory at 1313 N. Second Street, Phoenix. Historically, the latter was among the first of the post WW II "medical centers" which proliferated ad infinitum in Phoenix and ended the supremacy of the Professional Building, 15 E. Monroe and the Grunow Clinic on East McDowell, which were the principal centers of family practice and specialty foci in Phoenix.

Lee was a *doer* and a *booster* in ofessional and medical activities. A rtial listing is imposing. He succeeded ank Malloy and served several years as e second editor of *Arizona Medicine*; he rved as secretary of the medical staff of od Samaritan Hospital; state councilor r the Radiological Society of North nerica; councilor for the American llege of radiology for the State; mber of the American Radium Society, e American Roentgen Ray Society, F A R; member of the American College of ysicians, F A C P ; the county society, te and American Medical Associations d was appointed to the first Arizona omic Energy Commission.

So well did he balance his time, and all e while devoted to his family, that it s difficult to tell his vocation, diology, from his avocation, church ties, both local and national.

He became a proficient pilot and served staff radiologist to the Pinal General spital in Florence, the Hoemako ospital in Casa Grande and the Morenic spital in Morenci.

The general radiologist is on the wane d with Lee we have lost one of these neralists. To his devoted wife, Nelle d his family we extend our sincere mpathy.

W. Kennedy, M.D.
story and Obituaries Editor

ArMA Reports

E MINUTES APPEARING IN THIS SECTION HAVE EN EDITED TO CONSERVE SPACE. A COMPLETE PY OF THE MINUTES OF ANY MEETING WILL BE AILED TO ANY MEMBER REQUESTING THEM.

Executive Committee

The meeting of the Executive ommittee of the Arizona Medical ssociation, Inc. held in the Kudu Room f the Safari Resort, Scottsdale, AZ on uesday, April 24, 1979, a quorum being resent, convened at 10:01 p.m., Richard . Dexter, M.D., President and hairman, presiding.

Department of Health Services ctivity Report

Dr. Dandoy briefly updated several tems of general interest, including the act that a varied group of health rofessionals from throughout the state vere scheduled to meet during the week elative to the future of the Arizona Children's Hospital; the fact that a symposium is to be held within the next few days regarding the recent aflatoxin concern; and the fact that, due to the very obvious nurse shortage within the state, a group would be meeting in the very near future to determine what, if anything, could be done in an effort to recruit additional nurses.

Candidacy of Daniel T. Cloud, M.D.

The committee was advised that it was the intention of Daniel T. Cloud, M.D. to seek the presidency of the American Medical Association in 1980. Following discussion,

IT WAS MOVED AND CARRIED TO EDORSE THE 1980 CANDIDCY OF DANIEL T. CLOUD, M.D. FOR PRESIDENT OF THE AMERICAN MEDICAL ASSOCIATION.

Mailgram Action Bank

Following a review of the March 27, 1979 letter from the American Medical Association regarding a new communications tool known as "Mailgram Action Bank" and a brief statement by Dr. Nesbitt concerning the matter,

IT WAS MOVED AND CARRIED TO REFER THE MARCH 27, 1979 LETTER OF THE AMERICAN MEDICAL ASSOCIATION RELATIVE TO THE "MAILGRAM ACTION BANK" TO THE LEGISLATIVE COMMITTEE AND TO THE ARIZONA MEDICAL POLITICAL ACTION COMMITTEE FOR THEIR FURTHER INVESTIGATION, REVIEW AND ACTION, AS DEEMED NECESSARY.

Review of Board of Directors' Agenda for 4/25/79

The committee reviewed the agenda for the Board meeting April 15, 1979, with additions and changes being made.

Correspondence

Letter dated March 21, 1979 from Governor Bruce Babbitt re Board of Medical Examiners appointments. RECEIVED.

Letter and memo dated April 10 and April 18, 1979, respectively from the American Medical Association relative to chiropractic litigation. RECEIVED.

BOARD OF DIRECTORS

The meeting of the Board of Directors of the Arizona Medical Association, Inc. held in the Frech Quarter of the Safari Resort, Scottsdale, Arizona, on Wednesday, April 25, 1979, a quorum being present, convened at 8:02 a.m., Richard L. Dexter, M.D., President and Chairman presiding.

BOARD OF DIRECTORS

Suggested 1979-80 Meeting Schedule

IT WAS MOVED AND CARRIED TO APPROVE THE FOLLOWING SCHEDULE OF BOARD OF DIRECTORS' MEETINGS: AUGUST 4, 1979 -SATURDAY, 10:00 A.M.; OCTOBER 6, 1979 - SATURDAY, 10:00 A.M.; JANUARY 5, 1980 - SATURDAY, 10:00 A.M.; MARCH 8, 1980 - SATURDAY, 10:00 A.M.; APRIL 30, 1980 - WEDNESDAY, 8:00 A.M. - ANNUAL MEETING: AND MAY 2, 1980 - FRIDAY - 12:00 NOON - ANNUAL MEETING.

BOMEX Lease Extension

IT WAS MOVED AND CARRIED TO ACCEPT THE LEASE EXTENSION WITH THE BOARD OF MEDICAL EXAMINERS FOR THE PERIOD 8/1/79 to 7/30/80 FOR A PORTION OF THE ARIZONA MEDICAL ASSOCIATION BUILDING LOCATED AT 810 WEST BETHANY HOME ROAD, PHOENIX, ARIZONA, FOR AN ANNUAL RENTAL OF $16,386.50, COMPUTED AT THE RATE OF $6.50 PER SQUARE FOOT.

EXECUTIVE COMMITTEE

Membership Classification Changes Approved

Maricopa County Medical Society

1. James A. Austin, M.D. - Active to Associate - Account Military Service - Dues Exempt - Effective 1/1/79.
2. Earl M. Lustgarten, M.D. - Active to Active Over 70 -Account Age - Dues Exempt - Effective 1/1/79.
3. Robert T. Phillips, M.D. - Active to Active over 70 -Account Age - Dues Exempt - Effective 1/1/79.
4. E. Henry Running, M.D. - Active to Active Over 70 -Account Age - Dues Exempt - Effective 1/1/79.
5. M. W. Merrill, M.D. - Active to Active Over 70 -Account age - Dues Exempt - Effective 1/1/79.

Pima County Medical Society

1. Miguel A. Carreras, M.D. - Active to Associate -Account Retirement - Dues Exempt - Effective 1/1/79.
1. Merlin K. DuVal, M.D. - Active to Affiliate - Account Out-of-State - Dues Exempt - Effective 1/1/79.
3. Eugene Hildebrand, M.D. - Associate - Account Retirement - Dues Exempt - Effective 2/13/79.
4. Farooq Jaffer, M.D. - Active to Affiliate - Account Out-of-State - Dues Exempt - Effective 1/1/79
5. John G. Kloss, M.D. - Active to Associate - Account Residency - Dues Exempt - Effective 1/1/79.
6. Raymond F. Oyler, M.D. - Active to Associate - Account Retirement - Dues exempt - Effective 1/1/79.
7. Nagi K. Saied, M.D. - Active to Associate - Account Residency - Dues Exempt - Effective 1/1/79.

1980 Candidacy of Daniel T. Cloud, M.D.

The Board was informed that Daniel T.

Cloud, M.D., had recently announed his intention to seek the office of President of the American Medical Association in 1980. Following a discussion,

IT WAS MOVED AND CARRIED THAT THE ARIZONA MEDICAL ASSOCIATION ENDORSE AND ACTIVELY SUPPORT THE CANDIDACY OF DANIEL T. CLOUD, M.D. FOR PRESIDENT OF THE AMERICAN MEDICAL ASSOCIATION IN 1980.

ECONOMIC ORGANIZAITONS FOR PHYSICIANS, AD HOC COMMITTEE ON

The Board reviewed the action taken by the Ad Hoc Committee on an Economic Organization for Arizona Physicians at its meeting on January 6, 1979; and, following a lengthy discussion of the matter,

IT WAS MOVED AND CARRIED THAT THERE BE AN ECONOMIC ORGANIZATION FOR ARIZONA PHYSICIANS AND THAT SUCH AN ORGANIZATION BE A PART OF THE ARIZONA MEDICAL ASSOCIATION FOR THE PURPOSE OF NEGOTIATING CONTRACTS, CARRYING ON INSURANCE GRIEVANCES FOR ITS MEMBERS, AND MONITORING THIRD PARTIES: AND THAT THIS ACTION BE REPORTED TO THE HOUSE OF DELEGATES IN MEETING ON APRIL 27, 1979.

FINANCE COMMITTEE

Audit Report

Audit Report of Henry & Horne, Certified Public Accountants, for the year 1978. RECEIVED.

Financial Statement

Financial Statement for period ending 3/31/79 RECEIVED.

Membership Count

Memership Recap by County Society and Class of Membership as of 4/30/79 RECEIVED.

LEGISLATIVE COMMITTEE

1979 Legislative Status Report

Mr. Gary L. Barnett submitted to the Board a copy of the Annual Report of the Legislative Committee setting forth ultimate action taken in the legislature during the 1979 session concerning matters of interest to the Arizona Medical Association.

MEDICAL EDUCATION COMMITTEE

CME Hospital Accreditations

Accreditations granted to Camelback Hospital, St. Luke's Hospital and Phoenix Baptist Hospital RECEIVED.

AMA DELEGATION

Recommendations for AMA Appointments to councils and Committees

IT WAS MOVED AND CARRIED TO PLACE THE REQUEST OF THE AMERICAN MEDICAL ASSOCIATION DATED JANUARY 22, 1979 FOR RECOMMENDATIONS FOR APPOINTMENTS TO AMA COUNCILS AND COMMITTEES ON THE AGENDA FOR THE BOARD OF DIRECTORS MEETING SCHEDULED FOR APRIL 27, 1979, AND, ADDITIONALLY, TO REFER A COPY OF SUCH REQUEST TO THE MEDICAL EDUCAITON COMMITTEE FOR SUCH ACTION AS IT DEEMS APPROPRIATE.

Candidates for AMA Elective Positions

Letter of the American Medical Association dated 4/9/79 RECEIVED.

CORRESPONDANCE

Letter of 3/30/79 from the American College of Emergency Physicians RECEIVED.

Letter of 3/7/79 from the American Association of Medical Assistants, Inc. RECEIVED.

Letter of 2/20/79 from the Arizona Medical Association Auxiliary, Inc. RECEIVED.

OTHER BUSINESS

Professional Committee

Standards for Free Standing Emergency Facilities

The Board reviewed and considered the recommendation of the Professional Committee dated April 17, 1979, as it related to minimum standards for free standing emergency medical facilities and, following discussion,

IT WAS MOVED AND CARRIED THAT THE ARIZONA MEDICAL ASSOCIATION ENDORSE THE STANDARDS FOR MINIMAL REQUIREMENTS FOR FREE STANDING EMERGENCY FACILITIES ADOPTED BY THE ARIZONA CHAPTER, AMERCIAN COLLEGE OF EMERGENCY PHYSICIANS IN SEPTEMBER 1978; AND THAT THOSE STANDARDS, TOGETHER WITH THE POSITION TAKEN BY THE BOARD OF DIRECTORS OF ARMA, BE DISSEMINATED TO ALL APPROPRIATE AGENCIES.

Biofeedback & Advertising

IT WAS MOVED AND CARRIED THAT THE RECOMMENDATIONS OF THE PROFESSIONAL COMMITTEE MADE DURING ITS MEETING OF APRIL 3, 1979 RELATIVE TO BIOFEEDBACK AND ADVERTISING BE TABLED.

BOARD OF DIRECTORS

The meeting of the Board of Directors of the Arizona Medical Association, Inc., held in the Lanai room of the Safari Resort, Scottsdale, Arizona on Friday, April 27, 1979, a quorom being present, convened at 12:01 p.m., William E. Crisp, M.D., President and Chairman, presiding.

COMMITTEE CONFIRMATIONS

IT WAS MOVED AND CARRIED TO CONFIRM THE APOINTMENTS TO THE FOLLOWING COMMITTEES:

Note: The single asterisk indicates reappointment. The double asterick indicates new appointments. Those without astericks are previous year appointments whose terms are still in effect. They are listed here for information only.

ARTICLES OF INCORPORATION AN BYLAWS COMMITTEE - TERM 1979-80.

*C. E. Henderson, M.D., Chairman
J. A. Ash, M.D., Secretary
**E. P. Brown, M.D.
J. T. Clymer, M.D., President-Elect
W. E. Crisp, M.D., President
*P. E. Dew, M.D.
*Paul B. Jarrett, M.D.
**J. K. Kerr, M.D.
*W. D. Rummel, M.D.
*E. Sattenspiel, M.D.
*W. C. Scott, M.D.
**N. O. Ward, M.D.
W. F. Wrothen, M.D.

ArMPAC BOARD OF DIRECTORS - TER 1979-80

**J. F. Kahle, M.D., Chairman
**L. Anderson, M.D.
J. A. Ash, M.D., Secretary
*Mrs. L. P. Carter
J. T. Clymer, M.D., President-Elect
W. E. Crisp, M.D., President
**R. Dale, M.D.
**K. E. Durfee, M.D.
**Mrs. R. Goodsite
*R. L. Hagan, M.D.
*Mrs. C. W. Johnson
*D. V. Langston, M.D.
**D. W. Longfellow, M.D.
*D. T. Meredith, M.D.
*T. F. Moore, M.D.
Mrs. J. H. Reno
*J. H., Vosskuhler, M.D.
*Mrs. R. H. Wohl
**Mrs. H. Wuesthoff

AWARDS COMMITTEE, AD HOC - TER 1979-80

**R. L. Dexter, M.D., Chariman
J. A. Ash, M.D., Secretary
J. T. Clymer, M.D., President-Elect
W. E. Crisp, M.D., President
**J. F. Kahle, M.D.
**E. Sattenspiel, M.D.

MALPRACTICE INSURANCE CRISIS COMMITTEE AD HOC - TERM 1979-80

*E. Sattenspiel, M.D., Chairman
J. A. Ash, M.D., Secretary
*L. M. Brickler, D.O.
J. T. Clymer, M.D., President-Elect
W. E. Crisp, M.D., President
**V. F. Lovett, M.D.
*W. H. Magen, M.D.
*A. R. Nelson, M.D.
*D. F. Schaller, M.D.
*J. J. Standifer, M.D.
**D. S. Trump, M.D.
**J. Wetherell, M.D.
**A. Yudell, M.D.

BENEVOLENT AND LOAN FUND COMMITTEE

	TERM
*A.V. Dudley, Jr., M.D., Chairman	1979-8 1979-82
J.A. Ash, M.D.,Secretary	1979-8
J.T. Clymer, M.D., President-Elect	1979-8
W.E. Crisp, M.D., President	1979-8
*D.C. DeBoer, M.D.	1979-82
R.L. Foster, M.D.	1977-8
*A.M. Goldner, Ph.D.	1979-82
P.N. Levin, M.D.	1978-81
G.S. Naifeh, Jr., M.D.	1978-81
N.L. Robles, M.D. Treasurer-Specified	1979-80
*C. C. Vaughn, Jr., M.D.	1979-82

FINANCE COMMITTEE

	TERM
.L. Robles, M.D., Chairman	1979-80
A. Ash, M.D., Secretary	1979-80
arl J. Baker, M.D.	1978-81
T.Clymer, M.D, President-Elect	1979-80
.E. Crisp, M.D., President	1979-80
erbert G. Erhart, Jr., M.D.	1978-81
. Goldsmith, M.D.	1977-80
L. Grobe, M.D.	1978-81
rry W. Haggard, M.D.	1977-80
W. Kennedy, M.D.	1977-80
.T. Maloney, M.D.	1977-80
.L. Mobley, M.D.	1979-82
.I. Shapiro, M.D.	1977-80
.S. Smith, M.D.	1979-82
.C. Tretbar, M.D.	1978-81

GOVERNMENTAL SERVICES COMMITTEE

	TERM
C. Kossuth, M.D.,Chairman	1979-80
	1977-80
A. Ash, M.D., Secretary	1979-80
.T. Clymer, M.D., President-Elect	1979-80
.E. Crisp, M.D., President	1979-80
. A. Davis, M.D.	1979-82
. Kalil, M.D.	1978-81
. D. Peters, M.D.	1979-82
.A. Reed, M.D.	1979-82
. I. Rosenblatt, M.D.	1977-80
. St. John, M.D.	1979-82
M.C. Schneider, M.D.	1977-80
.D. Zeitzer, M.D.	1978-81

GRIEVANCE COMMITTEE

	TERM
R.L. Dexter, M.D., Chairman	1979-80
.W. Abbuhl, M.D.	1979-82
.A. Ash, M.D., Secretary	1979-80
W.E. Berkley, M.D.	1977-80
.T. Clymer, M.D., President-Elect	1979-80
W.E. Crisp, M.D.	1979-80
P.E. Dew, M.D.	1978-81
M. H. Dubnow, M.D.	1978-81
A. H. Dysterheft, M.D.	1978-81
D. E. Glow, M.D.	1977-80
J.B. Jamison, M.D.	1978-81
J. N. Lane, M.D.	1977-80
R. T. McDonald, M.D.	1979-82
A. J. Ochsner, M.D.	1977-80

HEALTH MANPOWER COMMITTEE

	TERM
L. M. Linkner, M.D., Chairman	1979-80
	1979-82
J.A. Ash, M.D., Secretary	1979-80
J.T. Clymer, M.D., President-Elect	1979-80
W.E. Crisp, M.D., President	1979-80
B. N. Curtis, M.D.	1977-80
J. A. Dupont, M.D.	1979-82
H. V. Grumbling, Jr., M.D.	1979-82
T. H. Hicks, M.D.	1978-81
L.C. Kossuth, M.D.	1978-81
M. M. Marble, M.D.	1979-82
W. J. Nieri, M.D.	1978-81
S.L. Posner, M.D.	1977-80
M.I. Roth, M.D.	1977-80
G. B. Rowland, M.D.	1978-81
R. P.Spark, M.D.	1978-81
A.F. Vuturo, M.D.	1977-80

HISTORY AND OBITUARIES COMMITTEE

	TERM
M. B. Block, M.D., Chairman	1979-80
J.A. Ash, M.D., Secretary	1979-80
J.T. Clymer, M.D., President-Elect	1979-80
W.E. Crisp, M.D., President	1979-80
J.R. Harlan, M.D.	1978-81
J. W. Kennedy, M.D.	1977-80
W. D. Rummel, M.D.	1977-80
W. C. Weese, M.D.	1977-80

LEGISLATIVE COMMITTEE

	TERM
**R.O. Flynn, M.D., Chairman	1979-80
	1978-81
J.A. Ash, M.D.,Secretary	1979-80
**W. E. Beck, M.D.	1979-82
**D. Bright, M.D.	1979-82
R.G. Bubeck, D.O.	1977-80
J. E. Campbell, M.D.	1977-80
*W.S. Chisholm, Jr., M.D.	1979-82
J.T. Clymer, M.D., President-Elect	1979-80
*D. V. Cookro, M.D.	1979-82
W.E. Crisp, M.D., President	1979-80
**W. J. Dooley, M.D.	1979-82
K.A. Dregseth, M.D.	1978-81
T. F. Griffin, Jr., M.D.	1978-81
J. M. Hurley, M.D.	1977-80
G. Lastnick, M.D.	1978-81
R. McGill, D.O.	1978-81
**J. B. McNally, M.D.	1979-82
D. R. Miles, M.D.	1977-80
T. F. Moore, M.D.	1978-81
D. A. Ogden, M.D.	1978-81
*R.M. O'Harra, M.D.	1979-82
K. L. Pomeroy, M.D.	1977-80
W. M. Potter, M.D.	1977-80
**J. S. Pucelick, M.D.	1979-82
**R. D. Sanford, M.D.	1979-82
E. Sattenspiel, M.D.	1977-80
**J.L. Schamadan, M.D.	1979-82
B.S. Strug, M.D.	1978-81
**W. A. Susong, M.D.	1979-82
J.W. Vosskuhler, M.D.	1978-81
Neil White	1977-80

MATERNAL AND CHILD HEALTH CARE COMMITTEE

	TERM
*W.C. Scott, M.D., Chairman	1979-80
	1979-82
J.A. Ash, M.D., Secretary	1979-80
F.W. Baum, M.D.	1978-81
*J.F. Blute, III, M.D.	1979-82
G.L. Busch, M.D.	1978-81
W.B. Cherny, M.D.	1977-80
J.T. Clymer, M.D., President-Elect	1979-80
W.A. Colton, Jr., M.D.	1978-81
O.L. Cranmer, M.D.	1977-80
W.E. Crisp, M.D., President	1979-80
W.J.R. Daily, M.D.	1977-80
J.A. Demlow, M.D.	1978-81
**W. Droegemueller, M.D.	1979-82
H.D. Gabel, M.D.	1978-81
J.M. Gillette, M.D.	1978-81
R.J. Jennett, M.D.	1977-80
J.V. Kelly, M.D.	1977-80
S. Kower	1978-81
M.K. Laughead, M.D.	1977-80
W.K. Lippard, M.D.	1978-81
G.T. McMahon, M.D.	1978-81
*H. B. P. Meyer, M.D.	1979-82
*Sister A. Montgomery	1979-82
*W. J. Moore, M.D.	1979-82
W. G. Payne, M.D.	1978-81
H. S. Rhu, Jr. M.D	1978-81
**L.S.Shenker, M.D.	1979-82
J.M. Shwayder, M.D.	1978-81
*W.A. Susong, M.D.	1979-82
P.A. Whitmore, D.O.	1977-80
**J.H. Wilson, M.D.	1979-82

MEDICAL ECONOMICS COMMITTEE

	TERM
*R.W. Switzer, M.D., Chairman	1979-80
	1979-82
**S.D. Abate, M.D.	1972-82
M.M. Abeshouse, M.D.	1977-80
J.A. Ash, M.D., Secretary	1979-80
R.S. Armstrong, M.D.	1978-81
C.G. Bennett, M.D.	1977-80
J.T. Clymer, M.D., President-Elect	1979-80
S.C.Colachis, M.D.	1977-80
W.E.Crisp, M.D., President	1979-80
*C.F. Dalton, M.D.	1979-82
W.M.Davis, M.D.	1978-81
**J.L. Frey, M.D.	1979-82
D.B. Gurland, M.D.	1977-80
A.D. Jacobson, M.D.	1978-81
F.W. Jensen, M.D.	1978-81
J.F.Kahle, M.D.	1978-81
L.M. Linkner, M.D.	1978-81
P.P. Moraca, M.D.	1977-80
R.W. Weber, M.D.	1978-81
**W.R. Womack, M.D.	1979-82
R.J.M. Zeluff, M.D.	1978-81

MEDICAL EDUCATION COMMITTEE

	TERM
*R.E.T. Stark, M.D., Chairman	1979-80
	1978-81
**L.A. Asadourian, M.D.	1979-81
J.A. Ash, M.D., Secretary	1979-80
J.E. Brady, Jr., M.D.	1978-81
J.T. Clymer, M.D., President-Elect	1979-80
W.E. Crisp, M.D., President	1979-80
*D. J. Crosby, M.D.	1979-82
*K.A. Dregseth, M.D.	1979-82
**P.C. Evans, M.D.	1979-82
F.T. Flood, M.D.	1978-81
W.S. Gold, M.D.	1977-80
S. Goldfein, M.D.	1978-81
S. M. Gould, Jr., M.D.	1978-81
*H.W. Hale, Jr., M.D.	1979-82
*R. E. Hastings, M.D.	1979-82
R.W. Hellmers, M.D.	1978-81
**P. Hernreid, M.D.	1979-82
W. Jacoby, Jr., M.D.	1977-80
J.M. Layton, M.D.	1977-80
D.J. Logue	1977-80
**J.R. Mouer, M.D.	1979-82
*H. N. Munhall, M.D.	1979-82
*A. Newman, M.D.	1979-82
**E.G. Ramsay, M.D.	1979-82
J. Schwimmer, M.D.	1978-81
**A.L. Serbin, M.D.	1979-82
W. F. Sheeley, M.D.	1977-80
*J.J. Standifer, M.D.	1979-82
L.Z.'Stern, M.D.	1978-81
A.B. Taylor, M.D.	1978-81
C. A. Trahern, M.D.	1977-80
A.F. Vuturo, M.D.	1978-81
A.G. Wagner, M.D.	1978-81
W. S. Williams, M.D.	1978-81
*A. C. Winfield, M.D.	1979-82

OCCUPATIONAL HEALTH COMMITTEE

	TERM
*J.M. Hughes, M.D., Chairman	1979-80 1977-80
J.A. Ash, M.D., Secretary	1979-80
F.K. Berk, M.D.	1978-81
E.M. Best, M.D.	1978-81
L.H. Boyea, M.D.	1978-81
W.F. Briney, M.D.	1977-80
J.T. Clymer,M.D., President-Elect	1979-80
W.E. Crisp, M.D., President	1979-80
R.B. Leonard, M.D.	1977-80
**R.L. Levitin, M.D.	1979-82
E.J. Ryan, M.D.	1977-80
**J.L. Schamadan, M.D.	1979-82
**E.W. Whiteford, Jr., M.D.	1979-82

PHYSICIAN REHABILITATION COMMITTEE

	TERM
*T.E. Bittker, M.D., Chairman	1979-80 1977-80
J.A. Ash, M.D., Secretary	1979-80
P.B. Borgesen, M.D.	1978-81
*H.G. Butler, M.D.	1979-82
J.T. Clymer, M.D., President-Elect	1979-80
R.E. Cotter, M.D.	1977-80
W.E. Crisp, M.D., President	1979-80
*E.W. Czerny, M.D.	1979-82
H.S. Gray, M.D.	1977-80
**T.S. Henry, II, M.D.	1979-82
B.R. Landess, M.D.	1977-80
L.M. Linkner, M.D.	1978-81
E.Meshorer, M.D.	1977-80
H.E. Pollock, M.D.	1977-80
*S.C. Scheiber, M.D.	1979-82
*W.B. Tomlinson, M.D.	1979-82
E.A. Waskow, M.D.	1977-80
M.A. Welleck, M.D.	1977-80
L.H. Westman, M.D.	1977-0
L.E. Young, M.D.	1977-80
V.J. Zannis, M.D.	1977-80

PROFESSIONAL COMMITTEE

	TERM
*J.W. Hanss, Jr., M.D., Chairman	1979-80 1978-81
J.A. Ash, M.D., Secretary	1979-81
P.M. Bindelglas, M.D.	1977-80
W.C. Brainard, M.D.	1978-81
J.T. Clymer, M.D. President-Elect	1979-80
W.E. Crisp, M.D., President	1979-80
J.A. Dupont, M.D.	1978-81
R.L. Hagan, M.D.	1977-80
J.M. Hurley, M.D.	1977-80
**D.B. Kunkel, M.D.	1979-82
C.W. Kurtz, M.D.	1977-80
C.R. Laing, M.D.	1978-81
*J.G. Lawson, M.D.	1979-82
W.L. Niccum, M.D.	1978-81
C.W. Pullen, M.D.	1978-81
**J.B. Scott, M.D.	1979-82
W.J. Thomas, M.D.	1978-81
W.R. Van Nostrand, M.D.	1977-80

PUBLIC RELATIONS COMMITTEE

	TERM
*I.B. Ehrlich, M.D., Chairman	1979-80 1979-82
R.B. Armstrong, M.D.	1977-80
J.A. Ash, M.D., Secretary	1979-80
M.B. Block, M.D.	1977-80
G.F. Brown, M.D.	1977-80
R.L. Christ, M.D.	1978-81
J.T. Clymer, M.D., President-Elect	1979-80
S.B. Choen, M.D.	1977-80
W.E. Crisp, M.D., President	1979-80
J. DeVries, Medical Editor	1978-81
S.E. Dippe, M.D.	1977-80
A.L. Gordon, M.D.	1978-81
A.A. Lilien, M.D.	1977-80
K.E. McReynolds, M.D.	1978-81
J.Schwimmer, M.D.	1977-80
**S. R. Stein, M.D.	1979-82

PUBLISHING COMMITTEE

	TERM
M.B. Block, M.D., Chairman	1979-80
J.T. Ash, M.D., Secretary	1979-80
F.K. Bagatel, M.D.	1977-80
B.R. Brown, M.D.	1978-81
J.T. Clymer, M.D., President-Elect	1979-80
W.E. Crisp, M.D., President	1979-80
W.V. Edwards, M.D.	1977-80
C.H. Fredell, M.D.	1978-81
**B.K. Harris, M.D.	1979-82
J.W. Kennedy, M.D.	1977-80
W.B. McGrath, M.D.	1978-81
J. Stoner, III, M.D.	1977-80
W C. Weese, M.D.	1977-80
*D.J. Ziehm, M.D.	1979-82

SCIENTIFIC ASSEMBLY COMMITTEE

	TERM
*J.M. Hurley, M.D., Chairman	1979-80 1977-80
*S.C. Anand, M.D.	1979-82
J.A. Ash, M.D., Secretary	1979-80
F.K. Berk, M.D.	1977-80
W.W. Bohnert, M.D.	1978-81
**G.E. Burdick, M.D.	1979-82
*W.S. Chisholm, Jr., M.D.	1979-82
J.T. Clymer, M.D., President-Elect	1979-80
W.E. Crisp, M.D., President	1979-80
*B.L. Crouch, M.D.	1979-82
J.A. Dupont, M.D.	1977-80
T.R. Harrington, M.D.	1978-81
T.F. Hartley, M.D.	1978-81
C.W. Kurtz, M.D. Vice President-Specified	1979-80
E. Leibsohn, M.D.	1977-80
P. Levy, M.D.	1977-80
*P. Mao, M.D.	1979-82
M.H. Nathan, M.D.	1977-80
W.S. Nevin, M.D.	1978-81
A. Newman, M.D.	1978-81
C. Richards	1978-81
N.L. Robles, M.D.	1977-80
*M.C. Schneider, M.D.	1979-82
**R.A. Sliver, M.D.	1979-82
D.P. Speer, M.D.	1978-81
Stephen R. Stein, M.D.	1977-80
L.Z. Stern, M.D.	1977-80
L.S. Tan, M.D.	1977-80
J.H. Targovnik, M.D.	1978-81
W.C. Voss, M.D.	1977-80
D.J. Ziehm, M.D.	1979-80

OTHER BUSINESS

Ad Hoc Committee on an Economic Organization for Arizona Physicians and on Health Care Cost

It was noted that the subject committees have been disbanded due to completion of assignments.

Ad Hoc Membership Committee

It was noted by the Board that this committee, as in the past, would consist of the several district directors, a representative of the Housestaff Section, a representative of the American Medical Student Association, as well as the President, President-Elect and Secreta of the Association.

Recommendations for AMA Appointments Councils and Committees •

The Board reviewed and considered t request of the American Medical Associati dated January 22, 1979 for submission recommendations for appointment to AN Committees and Councils. Followi discussion,

IT WAS MOVED AND CARRIED THAT TH ARIZONA MEDICAL ASSOCIATIO SUBMIT TO THE AMERICAN MEDICA ASSOCIATION BY MAY 10, 1979, TH NAMES OF THE FOLLOWING PHY ICIANS, RECOMMENDING THEI APPOINTMENT TO THE COMMITTEE COUNCILS IMMEDIATELY FOLLOWIN THEIR NAMES: EDWARD SATTENSPIE M.D. - RESIDENCY REVIEW COMMITTEE OBSTETRICS-GYNECOLOGY; RICHAR D. ZONIS, M.D. - RESIDENCY REVIE COMMITTEE -OTOLARYNGOLOG MAC DONALD WOOD, M.D. - RESIDENC REVIEW COMMITTEE - SURGERY; AN WILLIAM R MYERS, M.D. - RESIDENC REVIEW COMMITTEE - INTERNA MEDICINE AND LIAISON COMMITTE ON CONTINUING MEDICAL EDUCA TION.

IT WAS FURTHER MOVED AN CARRIED THAT ANY NAMES RECEIVE AT THE OFFICE OF ArMA PRIOR TO TH MAY 10, 1979 DATE WOULD LIKEWISE E SUBMITTED TO THE AMA WITH TH RECOMMENDATION OF THE ASSOCIA TION TO THE COMMITTEE/COUNC INDICATED ON THE RECOMMENDA TION SHEET.

MATERNAL AND CHILD HEALTH CARE COMMITTEE

The meeting of the Maternal and Chi Health Care Committe of the Arizor Medical Association, Inc. held at 810 We Bethany Home Road, Phoenix, Arizon on Saturday, May 5, 1979, convened 1:42 p.m., William C. Scott, M.D. Chairman, presiding.

LAY MIDWIFE PROGRAM

The Arizona Department of Healt Services letter dated April 3, 197 relating to an advisory committee for th lay midwife program was reviewed by th committee and Dr. Perry Stearr informed the membership that the fir meeting of the newly formed advisor committee was scheduled to be held i May.

Following a discussion relative t reporting forms on out-of-hospit deliveries,

IT WAS MOVED AND CARRIED T ACCEPT THE FORM RECOMMENDED BY THE ARIZONA DEPARTMENT OF HEALTH SERVICE ENTITLED "COMPLICATIONS REPORT, OUT-OF-HOSPITA DELIVERY", THAT THE HOSPITAL WITHIN THE STATE OF ARIZONA B INFORMED THAT SUCH A FORN WOULD BE FORTHCOMING FO

T EIR USE AND THAT, ALTHOUGH T IS WAS A VOLUNTARY EFFORT, I WAS THE RECOMMENDATION OF T IE MATERNAL AND CHILD ALTH CARE COMMITTEE THAT E CH HOSPITAL COOPERATE IN T IS NEW REPORTING SYSTEM.

RIZONA PERINATAL PROGRAM - LONG RANGE PLANNING

emo of H. Belton P. Meyer, M.D. ted 2/23/79. RECEIVED.

emo of Harold D. Gabel, M.D. dated 2/79. RECEIVED

atement of Raymond J. Jennett, M.D. CEIVED.

IPO GRANT

r. Gabel addressed the committee ative to the recently applied for proving Pregnancy Outcome Grant d the effect that such grant, if given, uld have on present or anticipated jects within the State of Arizona.

NDING FOR INDIGENT MATERN- Y AND INFANT PATIENTS IN RIZONA - PROPOSAL FOR CLUSION IN THE DEPARTMENT HEALTH SERVICES 1980-81 DGET.

r. Gabel spoke to the committee garding the care of indigent mother and ant patients in Arizona, presenting a le indicating losses presently sustained hospitals in delivering high risk thers, as well as a suggested phased ogram for funding for those patients d requested input from the committee mbers in order to timely prepare and bmit final figures for such a program to e Department of Health Services for clusion in its 1980-81 proposed budget.

CORRESPONDENCE

alth Systems Agency of Southeastern rizona

he letter of the Health Systems Agency Southeastern Arizona dated April 20, 79 regarding establishment of a project r pediatric services was reviewed by the mmittee and it was determined to refer e same to the Section on Services for hildren for further evaluation.

OTHER BUSINESS

EOC Guidelines on Pregnancy iscrimination Act

The committee received and reviewed e April 24, 1979 letter of Johnson & iggins setting forth the Guidelines on e Pregnancy Discrimination act cently issued by the Equal Employment pportunity Commission and it was etermined that the matter be referred to e section on Maternal Services for its nsideration and review as it might late to our presently pending legislation House Bill 2336.

At this point, the committee additionally scussed other matters of concern quiring some type of legislative action, cluding requirements for reporting of aternal mortalities within the state, quirements for certification of nurse practitioners, and it was the consensus of opinion that Richard O. Flynn, M.D., Chairman of the Legislative Committee, be requested to attend the next meeting of the Maternal and Child Health Care Committee, at which time an in-depth discussion of all of these matters might be held.

Genetics Services

Mr. Bob Perry, Executive Director of the March of Dimes, reported to the committee regarding a computerized system on birth defects which was now being made available through grants of $3,000.00 from the March of Dimes. Mr. Perry indicated that the grant was contemplated to cover the cost of installation of the system, as well as provide the basic initial operation. Following a discussion by Dr. Gabel regarding the need for information which would be available via such a system in determining genetic defects and aiding in delivery of high risk mothers.

IT WAS MOVED AND CARRIED THAT A SECTION ON GENETICS BE ESTABLISHED BY THIS COMMITTEE, THAT HAROLD D. GABEL, M.D. SERVE AS CHAIRMAN OF THAT SECTION; AND THAT A $3,000.00 GRANT FROM THE MARCH OF DIMES BE APPLIED FOR AND SUCH FUNDS, IF GIVEN, BE USED TO ESTABLISH THE TERMINAL FOR THE COMPUTERIZED SYSTEM ON BIRTH DEFECTS IN A CENTRALLY-LOCATED FACILITY TO SERVE THE ENTIRE STATE OF ARIZONA.

Perinatal Mobidity, Mortality & Peer Review

A request concerning the perinatal morbidity, mortality and peer review and the establishment of definitions of minimum standards in that regard was reviewed by the committee and it was determined that the matter should be referred to the committee's Section on Perinatal Planning for its consideration.

MEDICAL ECONOMICS

Meeting of the Medical Economics Committee of the Arizona Medical Association, Inc., held at 810 W. Bethany Home Road, Phoenix, Arizona, on Tuesday, May 22, 1979, convened at 7:22 p.m., Richard W. Switzer, M.D., Chairman, presiding.

INSURANCE PROGRAMS

The Chairman advised that very little has occurred since the last meeting with regard to the insurance package proposed by Mr. Russ Waters of the Southern California Agency. It was noted that the Committee may wish to solicit insurance package proposals from other agencies to be presented at a future meeting of the Committee. RECEIVED FOR INFORMATION.

PRESENTATION TO I.C.A. MEDICAL FEE SCHEDULE

The Committee was informed of the hearing of the Industrial Commission of Arizona for the purpose of reviewing the medical fee schedule on Thursday, May 24, 1979 at 9:00 a.m. It was noted that the fee schedule has not been revised since September 1, 1977 and is significantly lower than the usual and customary rates. There was considerable discussion on whether the Medical Economics Committee should make a presentation to the I.C.A. Drs. Switzer and Davis informed the Committee that the professional Guild of Arizona would be making a presentation to the Industrial Commission. The Guild has retained a labor managment expert, Mr. Herrick Roth, to make the presentation on their behalf. Mr. Roth has done extensive preliminary work on the presentation including talking with union leaders in Arizona and members of the Commission. The Guild is supporting a position of an on-going memorandum of understanding with the Commission. The specific fee schedule figures that the Guild would be requesting were not available. Considerable discussion ensued.

IT WAS MOVED AND CARRIED THAT THE MEDICAL ECONOMICS COMMITTEE SUPPORT THE PROFESSIONAL GUILD OF ARIZONA'S ATTITUDE WITH REGARD TO THE INDUSTRIAL COMMISSION OF ARIZONA AND TO RECOMMEND THAT THE ARIZONA MEDICAL ASSOCIATION SUPPORT THAT POSITION.

SCIENTIFIC ASSEMBLY COMMITTEE

The meeting of the Scientific Assembly Committee of the Arizona Medical Association, Inc. held at 810 W. Bethany Home Road, Phoenix, Arizona, on Saturday, June 2, 1979, convened at 12:43 p.m., James M. Hurley, M.D., Chairman, presiding.

ELECTION OF VICE-CHAIRMAN CHAIRMAN 1981 ANNUAL MEETING

Suresh C. Anand, M.D. was unanimously elected as Vice-Chairman of the committee, with the understanding that he will serve as chairman for the 1981 meeting.

FUTURE MEETINGS AND DATES

1980 - Registry Resort

Mr. Robinson addressed the committee relative to certain problems which had developed since the confirmation by the Registry Resort in Scottsdale for the holding of the 1980 Annual Meeting April 30 through May 4. Following a lengthy discussion, it was the consensus of opinion that negotiations would be entered into with The Pointe relative to the feasibility of the relocation of 1980's meeting from the Registry Resort to The Pointe. Should time and facilities prohibit such change for this year's meeting, it was determined to remain at the Registry Resort, accepting the space problem which has occurred.

1981 - The Pointe

Plans to hold the 1981 Annual Meeting of the Association April 22 through April 26 at The Pointe in Phoenix have been confirmed.

1982 Annual Meeting

It was determined that the Arizona Biltmore, the Camelback Inn and other hotels in the Phoenix/Scottsdale area would be considered for possible location of the 1982 Annual Meeting to be held April 28 through May 2.

CHOICE OF SCHOOL FOR 1981

After much discussion by the committee, a review of the letter received from the Maternal and Child Health Care Committee, as well as consideraton of the University of Arizona College of Medicine's offer to provide the 1981 program.

IT WAS MOVED AND CARRIED THAT, FOR THE 1981 ANNUAL MEETING, NO SPECIFIC MEDICAL SCHOOL WOULD BE CHOSEN TO PROVIDE CORE FACULTY FOR THE SCIENTIFIC SESSIONS BUT, ALTERNATIVELY, SPEAKERS WOULD BE INVITED ON A LOCAL LEVEL, REGIONAL AND NATIONAL LEVEL IN AN EFFORT TO PROVIDE SPEAKERS OF GEATEST RENOUN IN ALL FIELDS OF MEDICINE FOR A MORE INFORMATIVE AND EDUCATIONAL MEETING.

IT WAS ALSO MOVED AND CARRIED THAT, IN SELECTING SPEAKERS FOR THE 1981 MEETING, SPECIALTY SOCIETIES AND PHARMACEUTICAL COMPANIES WOULD BE CONTACTED FOR SUGGESTIONS AND THAT, WHILE THE BASIC FINANCING OF GUEST SPEAKERS WOULD BE ASSUMED BY THE ASSOCIATION, AID WOULD BE SOUGHT WHERE AN EXCESS AMOUNT MIGHT BE NECESSARY IN ORDER TO PROCURE ANY SPECIFIC SPEAKER.

REVIEW OF 1979 MEETING

Attendance Report of ArMA Members

Year Attend Memeber % Location

REVIEW OF 1979 MEETTING

Attendance Report of ArMA Members

Year	Attend	Members	%	Location
1979	603	2,688	22.4	Safari Resort - Scottsdale
1978	421	2,570	23.5	Mariott - Tucson
1977	498	2,556	16.5	Hyatt Regency - Phoenix
1976	704	2,471	28.5	Safari Resort - Scottsdale
1975	459	2,393	19.2	Braniff Hotel - Tucson
1974	660	2,232	29.6	Safari Hotel - Scottsdale
1973	654	2,079	31.5	Safari Hotel - Scottsdale
1972	550	1,866	29.5	Safari Hotel - Scottsdale
1971	390	1,745	22.4	Safari Hotel - Scottsdale
1970	337	1,669	22.6	Safari Hotel - Scottsdale

1979 Detailed Annual Meeting Report

The report, as presented, was discussed and it was the consensus of opinion that a more concentrated effort needed to be made to encourage the resident/intern segment of the medical profession to attend the annual meetings of the Association and that contact would be made with medical schools in this regard. Additionally, it was agreed that the appointment of a resident/intern to this committee would be helpful.

Review of 1979 Meeting Critique Forms

The committee reviewed the critique forms recently sent to the members of the Scientific Assembly Committee for their comments relative to the 1979 meeting and it was agreed that similar forms should be available for submission to *all* physicians attending the 1980 meeting in order to get more complete evaluation.

Attendance Report - Scientific Presentations

A report was submitted to the committee for its reivew regarding the number of attendees at each of the scientific presentations made during the meeting. RECEIVED.

Letter Received

Letters regarding the 1979 Annual Meeting received from Laurence M. Linkner, M.D., Louis E. Young, M.D., and Kathie DiCenso were reviewed by the committee. RECEIVED

Public Relations Report

The report of Owens & Associates was reviewed by the committee. RECEIVED

Commercial exhibitors

It was brought to the committee's attention that a member of the committee has made a positive gesture of thanks to all commercial exhibitors during this year's annual meeting and it was determined that, because the commercial exhibitors were such an improtant part of the success of the Association's annual meetings, each member of the committee should make every effort to meet and thank the exhibitors prior to the conlusion of each annual meeting.

Registration Fees

The registration fees for ann meetings were discussed, with general opinion being that the fees we in this day of inflation, a detriment good attendance.

IT WAS MOVED AND CARRIED RECOMMEND TO THE BOARD DIRECTORS TAHT $10.00 ASSOCIATION MEMBERSHIP DU BE ALLOCATED AND EARMARKI FOR ANNUAL MEETING EXPENS AND THAT THERE BE NO MEMBI REGISTRATION FOR THE SCIENTI FIC PORTION OF ArMA'S ANNU MEETING.

PLANS FOR THE 1980 MEETING

General Format

The general format, as approved by t Board of Directors, was discussed by t committee and the followir dterminations made:

ArMPA - In an effort to free up o evening, ArMPAC would be offered luncheon slot on Thursday as opposed the banquet held in the past.

Social activities - It was determin that, in addition to the golf and tenn tournaments, plans would be institut for the holding of a 5,000/10,0000 met race.

President's Reception - It was agre that the receptions for the ArM President and the ArMA Auxilia President would be held together prior the Presiden'S banquet.

Socio-Economic Program - Because the tremendous response to this year program, it was determined that a mo complete registration be kept on th segment of the annual meeting.

Specialty Society Participation

The committee reviewed letter received from the Arizona Society Otolaryngology and Maxillo-Faci surgery, the Arizona Society o Pathologists, the Arizona Thorac Society, the Society of Physical Medicin and Rehabilitation of Arizona and th Western Orthopedic Association RECEIVED.

veral of the Arizona specialty societies ng represented at the meeting, a gthy discussion was held regarding the es of the committee to bring about a ater degree of participation by these ieties in the annual meeting of the ociation, by the holding of meetings in junction with, bringing in speakers or simply by way of information to the mittee as to what particular subject y might desire to have included in the ntific presentations. The problems ting to this type of effort, i.e., istration fees, timing of meetings to incide, lack of enthusiasm due to type scientific presentations made, etc., re discussed.

veral of the specialty societies were remely receptive to a more involved ticipation, some having already mmitted to being included in the final nning and some indicating that letters ting forth their specific needs or ires in this regard would be thcoming.

pic Presented

e topics presented druing the years 76 through 1979 were briefly reviwed the committee. RECEIVED

akers and Topics

was determined that, in addition to st faculty form the University of lorado, the Ohio State Medical School uld be contacted regarding possible st speakers and that, until such lists ve been compiled, no specific topics ld be ascertained or decided upon. wever, it was noted that there was eptional interest expressed regarding munology/allergy, physical medicine d rehabilitation, organ transplants and diatrics.

he committee was also advised that, e to the cancellation of his physical ness presentation during 1979 Annual eting, Thomas E. Bittker, M.D. was re than willing to make this senation during the 1980 meeting.

cal Papers

ocal papers were discussed, with the mmittee receiving a breakdown of the mber offered and number used during e past three years. Following a cussion, it was agreed that a more ncentrated effort be made in soliciting pers from the residents and interns roughout the state and, as an added entive, a plaque procedure similar to at used for scientific exhibits be stituted.

ientific Program Worksheets

lthough no finalization regarding ogram worksheets could begin until the ation and dates of the 1980 Annual eeting have been ultimately termined, the committee received same r their information.

AUXILIARY PROGRAM PLANS

rs. Clymer, Mrs. Collins and Mrs. ewman briefly addressed the committee regarding the Auxiliary program for the 1980 Annual Meeting, indicating that, because of the several yet undetermined factors as far as location, etc., no specifics were available. The socio-economic program would follow the same theme, i.e., Physicians' Families, and hopefully would generate the same response; the tentative plan being for no guest speaker, but individual group sessions only, with discussion of varied subjects.

It was agreed that the Auxiliary would furnish the committee, a list of all meetings or social functions or luncheons which they need or want to hold during annual meeting in an effort to allow adequate room assignments.

It was also determined that the Auxiliary would be responsible for all necessary arrangements to be made in preparation for the newly-instituted 5,000/10,000 meter race.

PURCHASING COMMITTEE WESTERN PHYSICIANS PURCHASING ASSOCIATION

The meeting of the Purchasing Committee - Western Physicians Purchasing Association of the Arizona Medical Association, Inc. held at 810 West Bethany Home Road, Phoenix, Arizona, on Tuesday, June 5, 1979, convened at 7:20 p.m., John F. Kahle, M.D., Chairman, presiding.

WPPA - UPDATE

Products

Dr. Kahle advised the committee that Scherer had recently become Bergen-Brunswick Medical and a newly instituted policy was that all salesmen detailing doctors who presently are not members of WPPA must inform those physicians that they would obtain a better price on the same products by joining WPPA.

Additionally, new promotional flyers would soon be available with drug lists, pricing on office supplies, and an automobile flyer referring to the Chevrolet Citation.

Problems

Dr. Kahle commented at length about the various problems presently facing the WPPA, the greatest of which seemed to be financial. He reported that the total membership count to date was 849, comprised of 1,306 physicians. Ariozna's count, through June 5th, was 216 memberships; 377 physicians. Opinions were requested from the members as to how we might now best solicit membership of the remaining physicians in the state. It was the consensus that the best way was on a one-to-one basis and all members agreed to attempt this in their area. Everett Colbert, as a member of the Medical Group Mangers Association, agreed to solicit an invitation from that group to Dr. Kahle to speak to them on the benefits of the WPPA and, likewise, the President of the Arizona Chapter of the American Association of Medical would be contacted by the Committee in that regard; Ed Devalk indicated that he would author a letter to be used for promotional purposes setting forth the savings which had been realized by his Tucson group since joining and using WPPA; and it was determined that the next mailing should be sent stamped "Attention: Business Mangers" to see if there was any greater response.

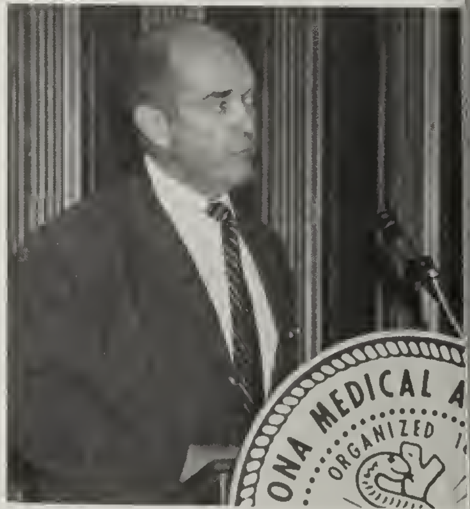
Annual Meeting Highlights

Past Presidents: seated L. to R. Drs. Harold W. Kohl, Abe I. Podolsky, Richard L. Dexter, Philip E. Dew, Edward Sattenspiel, William C. Scott. Standing Mr. Robert Carpenter, Drs. Dermont W. Melick, John J. Standifer, James E. O'Hare, W. Albert Brewer, Arthur V. Dudley, Clarence E. Yount, Richard O. Flynn, John F. Kahle, Carlos C. Craig, and Harry E. Thompson

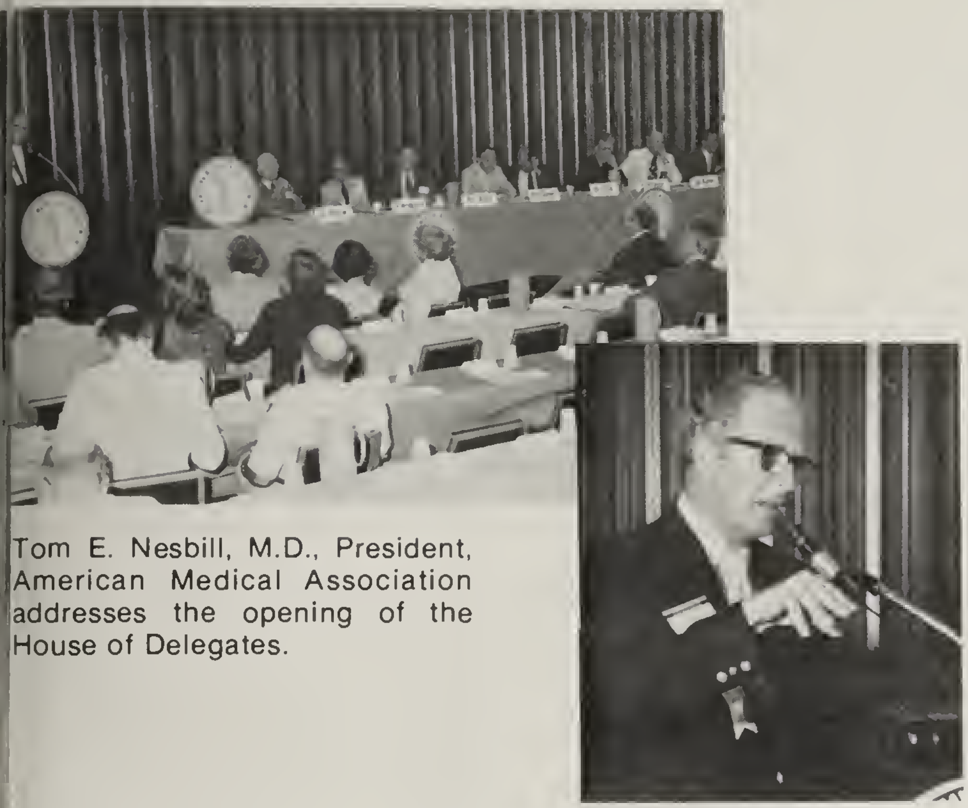
Daniel T. Cloud, M.
AMA Secy/Treas.

Richard L. Dexter, M.D., ArMA President 1978-79 & William E. Crisp, M.D., ArMA President 1979-80

Alan Gordon, M.D., President, Maricopa County Medical Society addresses the House of Delegates.



ArmA President William E. Crisp, M.D.



Tom E. Nesbill, M.D., President, American Medical Association addresses the opening of the House of Delegates.

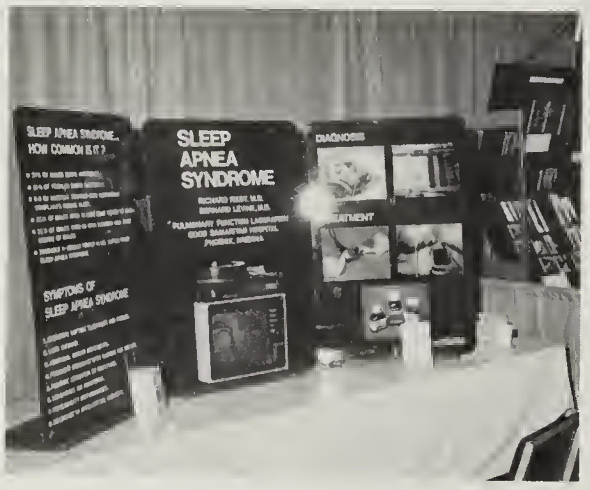
The 1979-80 ArMA Auxiliary officers
L. to R. Eloise Clymer, President; Joyce Newman, Pres.-Elect; Janet Hoffmann, First V.P.; Mary Eleanor Holmes, Treas.; and Carolyn Corrigan, Recording Secy.

Mrs. James L. Parsons (Carolyn) ArMA Auxiliary President 1978-79 addresses the House of Delegates.

Astronaut Ed Mitchell was the guest speaker at the President's banquet.

NEW DIRECTIONS

Betty Brazie and Kathie DiCenso admiring a few of th works for "Creative Moments '79."



Richard M. Riedy, M.D. and Bernard E. Levine, M.D. were winners with their scientific exhibit.

Dr. Kenneth Bistricky, Jim Allen and Dr. Kahle discuss the WPPA Program.

The two "Physician' Families" speakers Drs. Ellen and Perry Berman talk with Carolyr Parsons.

gnes Ash presents Dean Kettel with the Auxiliary's nnual contribution.

Tom E. Nesbitt, M.D., President, AMA.

Dr. Crisp presents Dr. Roy E. Burgess with his 50 Year Club award.

President's banquet 1979

NEW DIRECTIONS

The Arizona Medical Association, Inc. Resume'

of the

House of Delegates 1979 Annual Meeting

April 27, 1979

Adjourned meeting of the House of Delegates of the Arizona Medical Association, Inc. held in the Convention Center of the Safari Hotel, Scottsdale, Arizona, Friday, April 27, 1979, convened at 8:14 a.m., Robert A. Price, M.D., Speaker of the House, presiding.

Credentials—The Credentials Committee reported a quorum present and the House duly constituted.

Roll Call—On the roll call 109 Delegates (and/or Alternates) and 26 members of the Board of Directors were present.

Minutes—Minutes of the meeting of the House of Delegates held April 29, 1978, were approved as distributed.

Election of Officer—The following officers were elected for a term of one year.

President-Elect	J.T. Clymer, M.D.
Vice President	C. W. Kurtz, M.D.
Secretary	J.A. Ash, M.D.
Treasurer	N.L. Robles, M.D.
Speaker of the House	R.A. Price, M.D.
Editor-in-Chief	M.B. Block, M.D.

The following were elected for a term January 1, 1980 to December 31, 1981.

Delegate to AMA	E. Sattenspiel, M.D.
Delegate to AMA	W.S. Chisholm, M.D.
Alt. Delegate to AMA	B.N. Curtis, M.D.
Alt. Delegate to AMA	R.D. Zonis, M.D.

Election of District Directors The following District Directors were elected.

Central Dist. Dir.	R. Kirschner, M.D. (1979-1980)
Central Dist. Dir.	W.M. Potter, M.D. (1979-1982)
Central Dist. Dir.	J.K. Kerr, M.D. (1979-1982)
Northwestern Dist. Dir.	J.E. Oakley, M.D. (1979-1982)
Southeastern Dist. Dir.	K.A. Dregseth, M.D. (1979-1980)
Southern Dist. Dir.	J.S. Whaley, M.D. (1979-1982)

Reference Committee on Resolutions

Report of the Reference Committee on Resolutions as deleted, amended or as otherwise disposed of, adopted with the following actions taken on motions regularly made and carried.

Resolution 1-79

Subject: 1980 CALENDAR YEAR BUDGET OF INCOME AND EXPENSE

WHEREAS, It is customary for the House of Delegates to approve the Budget of Income and Expenditures for the next succeeding claendar year of the Arizona Medical Association, Inc.; therefore be it

RESOLVED, That the following Budget of Income and Expenditures for the calendar year 1980 be adopted:

	INCOME	EXPENDITURES	
General Dept.	$617,095.00	$681,100.00	$ (1,005.00
Committee Dept.	127,000.00	88,200.00	38,800.0
Annual Meeting Dept.	72,400.00	56,740.00	16,460.0
Publishing Dept.	78,170.00	77,982.00	188.0
Allocated-Future Projects	-0-	53,643.00	(53,643.00
TOTAL	$894,665.00	$894,665.00	$ -0

RESOLVED, That the annual dues of the Arizona Medical Association, Inc. be $250 for Active members, with $230 going to General Department Budget and $20 going to the Publishing Department Budget; and be it further

RESOLVED, That the Service member dues be $62.50, with $42.50 going to the General Department Budget and $20 going to the Publishing Department Budget; and be it further

RESOLVED, That the Intern or Resident Active member dues be $25, with $5 going to the General Department Budget and $20 going to the Publishing Department Budget.

ADOPTED 4/27/79

Resolution 2-79

Subject: P.L. 93-641; COMPOSITION OF HSA BOARD OF DIRECTORS

WHEREAS, P.L. 93-641 establishes the composition of the Board of Directors of Private Non-Profit Corporations which have been designated as Health Systems Agencies as follows:

1. "Consumers" (who must constitute a majority of the Board of Directors);
2. "Providers" (who must include one of each of the following: Health Professionals, Health Care Institutions, Health Care Insurance, Health Professional Schools, Allied Health Professionals, Private Health Agency, Veterans' Administration Representative, and HMO Representative who received more than 10% of their income from their medical activities); and
3. "Indirect Providers" as defined by Section 1531 (3) (B), as follows:

"Section 1531 (3) (B) who is an indirect provider of health care in that the individual—

(i) holds a fiduciary position with, or has a fiduciary interest in any entity described in subclause (II) or (IV) of clause (ii);

(ii) receives (either directly or through his spouse) more than one-tenth of his gross annual income from any one or combination of the following:

(I) Fees or other compensation for research into or instruction in the provision of health care.

(II) Entities engaged in the provision of health care or in such research or instruction.

(III) Producing or supplying drugs or other articles for individulas or entities for use in the provision of, or in research into, or instruction in the provision of health care.

(IV) Entities engaged in producing drugs or such other articles."

;and

WHEREAS, The "Indirect Providers" do not possess necessary medically-related education, experience or knowledge; and

WHEREAS, The "Indirect Providers" dilute the influence of the disignated "Providers" on that Board, to the detriment of the public interest; therefore, be it

RESOLVED, That the category of "Indir
Providers" be deleted; and, be it further
RESOLVED, That a resolution be introdu
to the American Medical Associati
recommending such action to the Congress
the United States.

**SUBSTITUTE RESOLUTION ADOPTE
4/27/79**

Resolution 3-79

Subject: NATIONAL HEALTH

WHEREAS, The American Medic
Association has adopted Resolution NO. 62
the House of Delegates' 1978 Interim Meetir
a
ndWHEREAS, The Arizona Medical
Association wishes to reaffirm its desire
maintain the effectiveness of the priva
health care system in the United Stat
therefore be it
RESOLVED, That the Arizona Medi
Association support the American Medi
Association's position which enbodies t
following principles:

1. Requiring minimum standards
adequate benefits in all health insuran
policies sold in the United States wi
appropriate deductible and co-insurance.

2. It is the responsibility of the state and/
local governments to provide medically f
those individuals who are unfortunate enou
(through no fault of their own, i.e., ag
disability, financial hardship, etc.) not to
able to provide for their own medical care.

3. A nationwide program by the priva
insurance industry of the United States (a
government if necessary for reinsurance)
make available catastrophic insuran
coverage for those illnesses and individu
where the economic impact of a catastroph
illness could be tragic. All catastroph
coverage should have an appropria
deductible and co-insurance to make
economically feasible and to avoid abuse.

4. A program developed pursuant to the
principles should be administered at the sta
level with national standardization throu
federal guidelines assuring the patients t
right to choose their own system of medi
care.

**SUBSTITUTE RESOLUTION ADOPTED A
AMENDED, 4/27/79**

Resoluiton 4-79

Subject: MEDICAL STUDENT SCHOLARSHIPS

WHEREAS, The Benevolent and Loan Fun
Committee has been given the responsibilit
for awarding $500.00 scholarships to worth
first year medical students who are Arizo
Residents; and
WHEREAS, All of these monies are derive
from interest earned by the general fund over
given calendar year; and

WHEREAS, The cost of living has tinuously gone up to the point where $)0.00 is only a very nominal amount; trefore, be it

RSOLVED, That, beginning in the year 1980, the Benevolent and Loan Fund Cmmittee be authorized to award solarships to first year medical students in t amount of $750.00 each.

ADOPTED AS AMENDED, 4/27/79

Rsolution 5-79

Sbject: THE AMERICAN COLLEGE OF OSTETRICIANS AND GYNECOLOGISTS' SATEMENT OF DECEMBER 1977 RGARDING ABORTION.

WHEREAS, The American College of Ostetricians and Gynecologists, in December 1977, issued a policy statement regarding aortion; therefore, be it

RSOLVED, That the following ten rommendations of the American College of Ostetricians and Gynecologists in their policy stement dated December 1977 be endorsed b the Arizona Medical Association.

. Education in family life, contraception, r ductive responsibility, and parenting sills.

2. Expansion of fundamental research in rproductive biology and behavior in order to povide better methods of family planning.

3. Removal of economic strictures to cmprehensive and quality health care svices, for example, by providing adequate pgnancy-related health coverage for all women.

4. Provision of adequate and supportive unseling presenting the practical ernatives for the management of problem pegnancies. Counseling directed solely ward either promoting or preventing aoriton does not sufficiently reflect the full ture of the problem or the range of options which the patient is entitled. Appropriately lanced counseling, combined with the ailable and accessible facilities, provides the nimum base for the opportunity to make a ly informed choice.

5. The elimination of practices which miss women from employment or deny em educational opportunities solely on the ounds of pregnancy.

6. The elimination of diability policies and actices which make it economically punitive continue a pregnancy.

7. The encouragement of research into nditions causing genetic or congenital nomalies and provision of better econceptual genetic counseling.

8. Encouragement of society to develop a ore supportive attitude toward the woman ho elects either abortion or an out-of-edlock birth.

9. Equal social, economic and medical nsideration for women who, once pregnant, hoose parenthood or adoption, as for those ho choose abortion. Adoption services hould facilitate this alternative where eferred.

10. Continuation of efforts to gather, ecord, and evaluate as objectively as possible he accumulating experience with aboriton and s alternatives in terms of their physical, sychological and social consequences, articularly their longterm consequences.

ADOPTED AS AMENDED, 4/27/79

Resolution 6-79

Subject: ROMAN G. MISLICKY SCHOLARSHIP

WHEREAS, Mr. Roman G. Mislicy was instrumental in helping the Arizona Medical Association initiate a Student Loan Program in coordination with the AMA-ERF in July 1967 to aid needy medical students in financing their medical education; and

WHEREAS, Mr. Mislicky has been instrumental in assisting new physicians in the State of Arizona to establish their practices through loan programs with local banks; and

WHEREAS, Mr. Mislicky has counseled many individual physicians so that they would not over-extend themselves financially to their future financial detriment; and

WHEREAS, Mr. Mislicky has been very active in the field of health care as a consumer member of many medically-oriented organizations; and

WHEREAS, Mr. Mislicky has been most helpful in counseling the Association in respect to deposits of capital on a short term basis, the interest from which has provided funds for the Association's medical scholarship program; therefore, be it

RESOLVED, That the Arizona Medical Association designate one scholarship each year to entering freshman medical students of the University of Arizona College of Medicine as the "Roman G. Mislicky Scholarship."

SUBSTITUTE RESOLUTION ADOPTED, 4/27/79

REFERENCE COMMITTEE ON AMENDMENTS

Report of the Reference Committee on Amendments as deleted, amended or as otherwise disposed of, adopted with the following actions taken on motions regularly made and carried.

(Note: All bracketed, italicized words represent deletions: all fully capitalized words are new material.)

Resolution A-1-79

Subject: VOLUNTARY MEMBERSHIP - ARIZONA MEDICAL ASSOCIATION

NOT ADOPTED, 4/27/79

RESOLUTION A-2-79

Subject: UNIFIED MEMBERSHIP - AMERICAN MEDICAL ASSOCIATION

NOT ADOPTED, 4/27/79

Meeting adjourned Sine Die at 11:27 a.m.

Awards

Merlin K. DuVal, M.D. received the 1979 Physician Award for Community Services

Roy E. Burgess, M.D. and Donald F. Hill, M.D. were inducted into the 50 Year Club.

Stuart Holtzman, M.D. won the low net in the Golf Tournament

Tennis Tournament winners were: A & B mixed doubles Dr. and Mrs. Larry Nilsen, C & D mixed doubles Dr. & Mrs. Frederick Fingerhut and the men's mixed doubles, Dr. Hollis Phillips and Dr. Philip Hipps.

The Exhibit Attendance Award went to Floyd Berk, M.D., Tucson.

The Outstanding Scientific Exhibit Award was won by Bernard E. Levine, M.D. and Richard M. Riedy, M.D. for their exhibit on "Sleep Apnea Syndrome: A Common Clinical Problem."

"Creative Moments '79": "The Best of Show" was awarded to Regina Moynahan for the sculpture "Boy on a Skateboard."

"The Most Popular Vote" was a painting by Rheta Sawrey, "High Country."

Future Medical Meetings

CONTINUING MEDICAL EDUCATION

The following institutions and organizations have been accredited for their continuing medical education programs by the Liaison Committee on Continuing Medical Education.

Arizona Heart Institute
Arizona Medical Association, Inc.
Arizona State Hospital, Phoenix
Camelback Hospital, Phoenix
Desert Samaritan Hospital, Mesa
Good Samaritan Hospital, Phoenix
Health Maintenance Associates, Phoenix
Maricopa County General Hospital, Phoenix
Mesa Lutheran Hospital, Mesa
Phoenix Baptist Hospital & Health Center
Phoenix Indian Medical Center
Phoenix Memorial Hospital
Phoenix Psychiatric Council
St. Joseph's Hospital & Medical Center, Phoenix
Barrow Neurological Institute, Phoenix
St. Lukes's Hospital & Medical Center, Phoenix
Scottsdale Memorial Hospital
Tucson Hospitals Medical Education Program, Tucson
Veterans Administration Hospital, Phoenix
Veterans Administration Hospital, Prescott
University of Arizona College of Medicine, Tucson

The accredited Institutions and organizations above produce a variety of continuing medical education programs. Each accredited institution and organization is responsible for designating which of these programs meet ArMA's requirements for Category 1 credit. Physicians who participate in programs which are designated Category 1 by accredited institutions will receive Category 1 credit toward the ArMA Certificate in CME and the AMA's Physician's Recognition Award.

JULY

22nd Annual Ruidoso Family Practice Seminar
July 16-19, 1979. Inn of the Mountain Gods, Mescalero, New Mexico. Sponsor: New Mexico Chapter of the American Academy of Family Physicians. Contact: Bob Reid, Chairman, P.O. Box 456, Sunland Park, N.M. 88063. Approved for CME credit hours.

Advanced Cardiac Life Support Program - Provider
July 20 & 21, 1979. Arizona Medical Association offices, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Sponsor: Arizona Affiliate of the American Heart Assoc. Contact: Carol Turner, ArMA Offices, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

Summer Medical Seminar
July 28-29, 1979. Little America, Flagstaff, Az. Sponsor: The Coconino County Medical Society. Contact: Thomas Davee, M.D., 710 N. Beaver St., Flagstaff, AZ 86001. Approved for 8 hours Category 1 credit.

A Day of Surgery
July 27, 1979. Little America, Flagstaff, Az. Sponsor: The Arizona Chapter of the aemrican College of Surgeons. Contact: Herbert Fredell, M.D., 120 W. Fine Ave., Flagstaff, AZ 86001. Phone: (602) 774-2491. Approved for 5 hours of Cateory 1 credit.

AUGUST

ACLS Instructor Course
August 17 & 18, 1979. Arizona Medical Assoc. Office, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Sponsor: Arizona Affiliate of the American Heart Assoc. Contact: Carol Turner, ArMA Offices, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 16 hours category I credit.

Rocky Mountain Cancer Conference
August 17-19, 1979. Broadmoor Hotel, Colorado Springs, Co. Sponsor: American Cancer Society Colorado Medical Society & Colorado Society of Osteopathic Physicians. Contact; Rocky Mountain Cancer Conference. 1809 E. 18th Ave., Denver, Co. 80218.

SEPTEMBER

ACLS Recertification Course
September 21, 1979. Arizona Medical Association offices, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Sponsor: Arizona Affiliate of the American Heart Assoc. Contact: Carol Turner, ArMA offices, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

OCTOBER

Eighth Annual Symposium of the Western Division of the American Geriatrics Society. Management of Vascular Disease in the Elderly
October 26-27, 1979. Lakes Club, Sun City Arizona. Sponsor: University of Arizona College of Medicine and Walter O. Boswell Memorial Hospital. Contact: Julius Schwimmer, M.D., 13220 105th Ave. Bldg. B, Rm. 61, Sun City, AZ 85351. Approved for 11 hours of category 1 credit toward ArMA & AMA certificate. AAFP hours pending approval.

NOVEMBER

Southern Medical Association Annual Scientific Assembly
Las Vegas, Nevada, November 4-7, 1979, Sponsor: Southern Medical Assoc., & AMA. Contact: AMA Dept. of Meeting Services, 535 N. Dearborn St., Chicago, ILL. or Southern Medical Assoc., 2601 Highland Avenue, Birmingham. Alabama 35205.

27th Annual Meeting of the Medical Society of the United States and Mexico
November 14-17, 1979. Doubletree Inn, Tucson, Az. Sponsor: Medical Society of the United States and Mexico. Contact: Mrs. Carolyn Parsons, Executive Secretary, 3161 N. Pantano Road, Tucson, AZ 85715.

1980

FEBRUARY

Second Annual International Catara
Surgery Sumposium
February 18-20, 1980. Sponsor: Arizor
Fundation for Ophthalmic Research ar
Education. Contact: Ronald W. Barnet, M.D
Medical Director, 421 N. 18th St., Ste 10
Phoenix, AZ 85006. (602) 257-107
Approved for 18 hours of category 1 credi

MONTHLY OR WEEKLY

Shrine Medics Meeting
Second Tuesday of each month, Docto
Hospital, 5:45 p.m. J. South Classroo
Sponsor: Shrine Medics. Contact: Robert
Briggs, M.D., 5121 N. Central Ave., Phoeni
AZ 85012.

Office Psychiatry for the Primary Provider
2nd Monday of month, 4811 N. 7th Stre
Phoenix, AZ. Sponsor: Arizona Health Pla
Contact: T.R. Bittker, M.D., Box 500
Phoenix, AZ 85010. Approved for category
credit.

Counter Transference Group
Weekly, Thurs. 8—10 p.m. Sponsor: Phoen
Psychiatric Council. Contact: James
Campbell, M.D., 5051 N. 34th St., Phoenix, A
85018. Approved for 2 hours category
credit.

Monthly Meeting of Tucson Radiologists
Last Tues. of month. Plaza Internationa
Tucson, AZ. Sponsor: U of A Medical Cente
Dept. of Radiology. Contact: Irwin
Freundlich, M.D., U of A Health Science
Center, Dept. of, Radiology, Tucson, A
85724. Approved for 2 hours category
credit.

Pediatric Grand Rounds
Tuesday 7:30-8:30 a.m in Phoenix: 1
Tues.—Phoenix Indian Hospital, 2nd Tues.-
Maricopa County Hospital, 3rd Tues.—Goo
Samaritan Hospital, 4th Tues.—St. Joseph
Hospital. Sponsor: Maricopa Count
Hospital (Phoenix Hospital's Affiliate
Pediatric Program). Contact: J. Kip
Charlton, M.D., 2601 E. Roosevelt, Phoeni
AZ 85008. Approved for 1 hour per sessio
category 1 credit.

Review of Forensic Pathology Current Case, Special Topics
Thursday, weekly, 11 a.m., 120 S. 6th Ave
Phoenix, AZ. Sponsor: Arizona Society o
Pathologists. Contact: H.H. Karnitschnig
M.D., 120 S. 6th Ave., Phoenix, AZ. Approve
for 1 hour per session category 1 credit.

ARIZONA STATE HOSPITAL

2500 E. Van Buren, Phoenix, AZ. 85008
Contact: Arnold L. Kendall, M.D.

.H. Psychiatric Grand Rounds
2 Wed., 1:00-2:00 P.M., J-6 Conf. Rm., ntact: Dr. Conger & Staff
nical-Pathological Conference
3 Wed., 1:30-2:30 P.M. General Services lg., Conf. Rm.
dical Grand Rounds
4 Wed., 1:00-2:00 P.M., Medical Bldg. Conf.
ff Education Conference
ekly, Wed., 1 p.m., Contact: Howard E. lsin, M.D.

BARROW NEUROLOGICAL INSTITUTE

dical Education
rrow Neurological Institute of St. Joseph's spital and Medical Center, 350 W. Thomas ., Phoenix, AZ 85013. Sponsor: St Joseph's spital & Medical Center. Contact: John R. een, M.D. Approved for 1 hour category 1 edit.

urology Teaching Conference
esdays, 8:30-9:30 a.m., Eighth Floor Conf. om.

urosurgery Morbidity Conference
ednesdays, 8:15-9:15 a.m., on first and third eks, Eighth Floor Conference Room.

uro-ophthalmology Conference
esdays, 7:30 a.m. in 8th floor neurology nference room.

inal Injury Conference
ednesdays, 8:15-9:15 a.m., on second and urth weeks, in Neuropathology Conf. n.—a multidisciplinary review of admission neurosurgeons, 'orthopedists, and habilitation specialists

europathology of Gross Specimens onference
ursday, 7:30-8:30 a.m. in the Morgue.

eurology-Neurosurgical
linical-Neuroradiological and
europathological Conference
idays, 8-9 a.m., First Floor Conference Rm.

europathology or Neuroradiology onferences
riday, 9 a.m., Neuropathology in europathology Conference Rm., euroradiology in First Floor Conference m.

eurorehabilitation Conference
uesdays, noon, 8th Floor Conference Rm.

eurosurgical Journal Club
aturdays, 9-11 a.m. in Eighth Floor onference Rm.

CAMELBACK HOSPITAL

055 N. 34th St., Phoenix, AZ 85018. Sponsor: amelback Hospital. Contact: Medical irector.

Patient Staffing Conference
Three times weekly.

DESERT SAMARITAN HOSPITAL

1400 South Dobson Road, Mesa, Arizona, Contact: L.A. Rosati, M.D. Approved for category 1 credit. **Clinical Conference - Dept. of Medicine**
Weekly Thurs., 12:30-1:30 P.M. Buffet Lunch, Grande Rm.
Medical Education Programs
Wed., 7:00 P.M. Desert Samaritan Hospital, Mesa.

Monthly Visiting Professor Programs
Tuesday 7:00 P.M., Buffett 6:00 P.M.
OB/GYN Medical Staff Conference
Weekly, Monday, 12:30-1:30 P.M. Grande Rm.

Surgical Morbidity & Mortality Conference
2nd Thurs., 7:00 A.M., Grande Rm.

GOOD SAMARITAN HOSPITAL

1033 East McDowell Rd., Phoenix, AZ Contact: Warren A. Hinchcliffe, M.D. Approved for category 1 credit.

Anatomical Path. & Cytology
Weekly, Mondays 4:00-5:00 P.M., S-4 Conf Rm.
Gynecological Section Conference
2nd Monday, 12:30-1:30 P.M., West Rm
Monday Noon Conference
Weekly, 12:00-1:00 P.M. R-2 Conf. Rm.
Mortality/Morbidity, Surgery Comm.
2nd Monday, 11:30-2:00 P.M., West Rm.
Obstetrical Section Conference
1st Monday, 12:30-1:30 P.M., West Rm.
Pulmonary Disese Grand Rounds
Weekly, 12:00-1:00 P.M., D-5 North
Family Practice Conference
Weekly Tuesday, 12:00-1:00 P.M., Fam. Practice Conf. Rm.
Pediatric Grand Rounds
3rd. Tuesday, 7:300-8:30 A.M., West Rm.
Peri-Natal Mortality Conference
3rd Tuesday, West Rm. (May-August).
Surgical Grand Rounds
Weekly, Tuesday 7:00-8:30 A.M. East Rm.
Clinical Cancer Fourm
3rd Wednesday, 12:00-1:00 P.M., West Rm, Contact: John A. Bruner, M.D.
Combined Cardiovascular & Family Practice Conf.
Ist, 2nd, & 4th Wed., 12:00-1:00 P.M., D-5 North.
Dr. Finger's Lecture
Weekly, Wed., 12:30-1:30 P.M., R-2 Conf. Rm.
Learning Disabilities
Weekly, 10:30-12:30 A.M., R-2 Conf. Rm.
OBGYN Dept. Review & Statistics
Weekly, 12:30-3:00 P.M., OBGYN Conf. Rm.
PM&R Grand Rounds
1st. & 3rd. Wed., 12:00-1:00 P.M., R-2 Conf. Rm.
Gynecological Pathology
Weekly Thursday, 1:00-2:00 P.M., S-4 Conf. Rm.
GI Radiology Conference
1st Thursday, 12:00-1:00 P.M., F151, Radiology Conf. Rm.
Neurology Grand Rounds
Weekly Thursday, 9:00-10:00 A.M., D-5 North.
Tumor Treatment Planning Conf.
2nd & 4th Thursdays, 12:00-1:00 P.M., F151 Radiology
Joint Tumor Gyn Conference
2nd Friday, 12:30-1:30 P.M., West Rm.
Kinesology
Weekly Friday, 4:00-5:00 P.M., R-2 Conf Rm.
Medical Grand Rounds
Weekly Friday, 8:00-9:00 A.M., West Rm.
Neuropathy Conference
Last Firday, 1:15-2:15 P.M., S-4 Morgue.
Psychiatry Grand Rounds
Weekly Friday, 11:30-1:00 P.M. , East Rm.
Pediatric Wednesday Seminar
July, Sept., March 12:30-1:30 P.M., East RM.
Pediatric Thursday Seminar
May, June, July, 12:30-1:30 P.M., East Rm.
Phoenix Ultrasound Society Meeting
Sept., thru May, 2nd Thursday, 7-9:00 P.M., Med. Ed. Bldg. Contact: Dr. A. Ben-Ora.
PM&R Journal Club
Alt. Fridays, 12:00-1:00 P.M., R-2 Conf. Rm.
Family Practice Grand Rounds
1st. Wed.: April, Aug. & Dec., 7:30-9:00 A.M., West Rm.

MARYVALE SAMARITAN HOSPITAL

Continuing Medical Education Program
Every other Wednesday, 7-8 a.m.

KINO COMMUNITY HOSPITAL

2800 E. Ajo Way, Tucson, AZ 85713. Contact: Eric G. Ramsay, M.D. Approved for category 1 credit.

Attending's Hour
5th Monday, 12:30 P.M., Contact: Joseph Goldberger, M.D.
Infectious Disese Conference
3rd Monday, 12:30 P.M., Contact: Larry Lincoln, M.D.
Nephrology Conference
1st Monday, 12:30 P.M., contact: Alan Cohn, M.D.
OB/GYN Dept. Meeting
Weekly Mondays, 7:30 A.M., Contact: Lewis Shenker, M.D.
Psychiatric Conference
2nd & 4th Mondays, Contact: Jose Santiago, M.D.
Surgical Conference
Weekly Mondays, Contact: Ronald Fischer, M.D.
Cardiology Conference
4th Tuesday, 12:30 P.M., Contact: Brendan Phibbs, M.D.
Gynecology Conference
5th Tuesday, 12:30 P.M., Contact Lewis Shenker, M.D.
Neurology Conference
1st & 3rd. Tuesday, 12:30 P.M., Contact: H.W. Buschsbaum, M.D.
Radiology Conference
2nd Tuesday, 12:30 P.M., Contact: Marvin Baker, M.D.

Clinical Pharmacology
4th Wed., 12:30 P.M., Contact: Lee Gardner, Pharm.D.
Clinical Pathology Conf.
2nd Wed., 12:30 P.M., Contact: Philip Altman, M.D.
Gynecology Pathology Conference
Weekly Wed., 7:3'45 A.M., Contact: Lewis Shenker, M.D.
Medical Conference-Mortality & Morbidity
3rd Wed., 12:30 P.M., Contact: Joseph Goldberger, M.D.
Rheumatology Conference
1st Wed. 12;30 P.M., Contact: George Werner, M.D.
Cardiology Conference
1st Thursday, 12:30 P.M., Contact: Brendan Phibbs, M.D.
Clinical Correlation
3rd Thursday, 12:30 P.M., Contact: Elliot Munjack, M.D.
G-I Conference
4th Thursday, Contact: Ray Moldow, M.D.
OB/GYN Sonography/X-ray Conference
Weekly Thursday, 9:15 A.M., Contact: Lewis Shenker, M.D.
Chest Conference
2nd & 4th Fridays, 12:30 P.M., Contact: David Engelsberg, M.D.
Dermatology Conference
3rd Friday, 12:30 P.M., Contact: Peter Lynch, M.D.
Hematology Conference
1st Friday, 12:30 P.M., Contact: Steven Ketchel, M.D.

MARICOPA COUNTY GENERAL HOSPITAL

2601 E. Roosevelt, Phoenix, Az 85008. Contact: Steven Schaffner, M.D.

Medicine Conference
Daily 12:00-1:00 P.M., Contact: Steven Schaffner, M.D.
Chest Surgery Conference
Weekly Mondays, 1:30 P.M., Satan Cruz Rm.
Obstetrical Problem Conference
Weekly Tuesday, 7:15 A.M., Yavapai Rm
Orthopedic Conference
Weekly Tuesday, 7:30 A.M., Santa Cruz Rm.
Pediatric Grand Rounds
2nd Tuesday, 7:30-8:30 A.M.
Hand Surgery Conference
Weekly Wed., 7:30 A.M., Santa Cruz Rm.
Neurosurgery Discharge Planning
Weekly Wed., 1:30 P.M., Station 42.
OB/Neonatology Seminar
Weekly Wed. 7:15 A.M., Yavapai Room
Psychiatry Conference
Weekly Wed. 11:00-12:00 A.M.
Surgery Conference
Weekly Wed., 7:00-8:00 A.M.
Cardiac/Cardiology Conference
Weekly Thurs. 12:00 P.M., Yavapai Room.
OB/GYN Resident Conference
Weekly Thurs. 12:00 P.M., Yavapai Rm.

GYN, Endocrine Seminar
Weekly Friday, 12:30 P.M., Santa Cruz Rm.
GYN Grand Rounds
Weekly Friday, 1:30 P.M., Santa Cruz Rm.
OB/GYN Surgical Pathology Conf.
Weekly Friday, 7:00 A.M., Yavapai Rm.
Orthopedic X-Ray Conference
Weekly Friday, 7:30 A.M., Santa Cruz Rm.

PHOENIX BAPTIST HOSPITAL & MED. CENTER

6025 N. 20th Ave., Phoenix, AZ 85015. Contact: James L. Grobe, M.D. Approved for category 1 credit.

Medical & Surgical Conference
Weekly Monday, 12;00 Noon
Medical Conference
1st. & 3rd Tuesdays, 12:00 Noon.
Medical & Surgical Conference
Weekly Wed., 12:00 Noon.
Medical & Surgical Conference
Weekly Friday, 12:00 Noon.

PHOENIX INDIAN MEDICAL CENTER

4212 North 16th St. Phoenix, AZ 85016. Contact: Leland L. Fairbanks, M.D. Approved for category I credit.

Pediatric Grand Rounds
1st Tuesday, 7:30-8:30 A.M.
Clinical Staff Teaching conference (Family Practice)
Weekly Wed., 8:00-9:00 A.M., Conf. Rm. A.

PHOENIX MEMORIAL HOSPITAL

120 S. 7th Ave. Phoenix, AZ. 85036. Contact: CME office, Approved for Category 1 credit.

Monthly Medical Education Seminar
3rd Monday, 6:30 P.M., Kiva Conf. Rm.
Tumor Board Conference
Weekly Friday, 12:00 P.M., Kiva Rm., Contact: Howard Kimball, M.D.

SCOTTSDALE MEMORIAL HOSPITAL

7300 East 4th St., Scottsdale, AZ 85251. Contact: W. S. Williams, M.D. Approved for category 1 credit.

Family Practice Conference
1st Monday, 12:45 P.M.
Morbidity & Mortality Conference
2nd Monday, 12:45 P.M.
Medical Grand Rounds
3rd Monday, 12:45 P.M.
Neurology & Neurosurgery Conference
3rd Monday, 12:30 P.M., Doctors' Lounge
Internal Medicine-Clinical Pathological Conference
4th Monday, 12:45 P.M.
Pulmonary Medicine Conference
1st Tuesday, 12:30 P.M., Doctors' Lounge
Cardiology Conference
2nd Tuesday 12:30 P.M., Doctors' Lounge
Surgery Departmental Meeting
3rd Tuesday, 12:15 P.M., Doctors' Lounge
Thoracic & Vascular Surgery Conference
4th Tuesday, 12:30 P.M., Doctors' Lounge
Cardiopulmonary Conference (Didactic)
4th Tuesday, 12:30 P.M., Doctors' Loung
Peri-Natal Mortality & Morbidity Conf.
2nd Wed., 12:30 P.M., Doctors' Lounge
Trauma Conference
3rd Thurs., 12:30 P.M., Doctors' Lounge
Tumor conference
Last Thursday, 12:30 P.M., Doctors' Loun
Psychiatry & Behavioral Scienc Conference
1st Friday, 12:30 P.M., Doctors' Lounge
GI Medical-Surgical-Radiologic Conference
2nd Friday, 12:30 P.M., Doctors' Lounge
Uronephrology-Oncology
Last Friday, 12:30 P.M., Doctors' Lounge

ST. JOSEPH'S HOSPITAL

350 West Thomas Road, Phoenix, AZ 850
Contact: Joseph C. White, M.D.

OB/GYN Section Conference
3rd & 4th Mondays, 12:30-1:30 P.M., 1st Flo Conf. Rm.
Pediatric Grand Rounds
4th Tuesday, 7:30-8:30 A.M., Contact: J. Ki Charlton, M.D.

ST MARY'S HOSPITAL & HEALTH CENTER

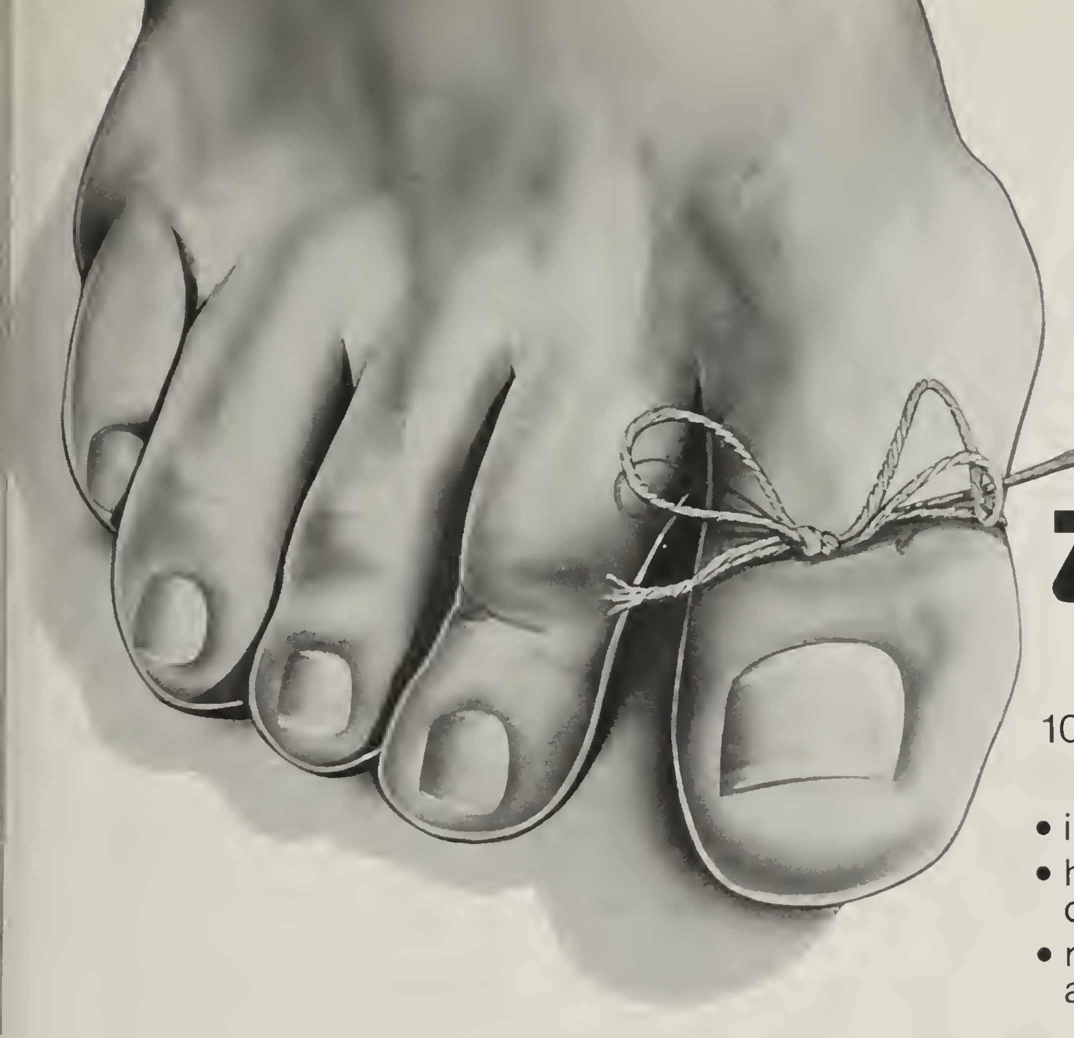
1601 W. St. Mary's Road, Tucson, AZ 8570
Contact: see below.

Monthly Specialty Conference-Dept. Surgery
1st Monday, 7:30 A.M., Century Rm. Contact: Med. Staff Office.
Grand Rounds: Medical Surgical, Famil Practice, Pathology, Radiology
Weekly Thursday
Cardiology Conference
Weekly Friday, 8:00-9:00 A.M., Century rm Contact: Anthony Forte, M.D.

U OF A HEALTH SCIENCES CENTER

Sponsor: U of A College of Medicine, Tucson AZ 85724. Contact: See below. Approved fo category 1 credit.

Anesthesiology Lectures
Weekly Thursday, 4:00-5:00 P.M.
Cardiac Catheterization Conference
Weekly Friday, 4:00 P.M., Contact: Dr Temkin.
Cardiology Research Conference
Weekly Tuesday, 7:30 A.M., Contact: Dr Roeske.
Case Discussion
Weekly Wed., 7:00 A.M., Din. Rm.C&D., Contact: Drs. Vaughan & Doyle.
Clinical Immunology, Allergy & Rheumatology Rounds
Every Friday, Noon-1 p.m. Contact: John Boyer, M.D., Dept. of Internal Medicine.



A reminder

ZYLOPRIM®
(allopurinol)
100 and 300 mg scored Tablets

- inhibits uric acid formation
- helps prevent urate crystal depositions in synovia
- reduces risk of uric acid lithiasis

INDICATIONS AND USE: This is not an innocuous drug and strict attention should be given to the indications for its use. Pending further investigation, its use in other hyperuricemic states is not indicated at this time.

Zyloprim® (allopurinol) is intended for:

1. treatment of gout, either primary, or secondary to the hyperuricemia associated with blood dyscrasias and their therapy;
2. treatment of primary or secondary uric acid nephropathy, with or without accompanying symptoms of gout;
3. treatment of patients with recurrent uric acid stone formation;
4. prophylactic treatment to prevent tissue urate deposition, renal calculi, or uric acid nephropathy in patients with leukemias, lymphomas and malignancies who are receiving cancer chemotherapy with its resultant elevating effect on serum uric acid levels.

CONTRAINDICATIONS: Use in children with the exception of those with hyperuricemia secondary to malignancy. The drug should not be employed in nursing mothers.

Patients who have developed a severe reaction to Zyloprim should not be restarted on the drug.

WARNINGS: ZYLOPRIM SHOULD BE DISCONTINUED AT THE FIRST APPEARANCE OF SKIN RASH OR ANY SIGN OF ADVERSE REACTION. In some instances a skin rash may be followed by more severe hypersensitivity reactions such as exfoliative, urticarial and purpuric lesions as well as Stevens-Johnson syndrome (erythema multiforme) and very rarely a generalized vasculitis which may lead to irreversible hepatotoxicity and death.

A few cases of reversible clinical hepatotoxicity have been noted and in some patients asymptomatic rises in serum alkaline phosphatase or serum transaminase have been observed. Accordingly, periodic liver function tests should be performed during the early stages of therapy, particularly in patients with pre-existing liver disease.

Patients should be alerted to the need for due precautions when engaging in activities where alertness is mandatory.

Nevertheless, iron salts should not be given simultaneously with Zyloprim. This drug should not be administered to immediate relatives of patients with idiopathic hemochromatosis.

In patients receiving Purinethol® (mercaptopurine) or Imuran® (azathioprine), the concomitant administration of 300-600 mg of Zyloprim per day will require a reduction in dose to approximately one-third to one-fourth of the usual dose of mercaptopurine or azathioprine. Subsequent adjustment of doses of Purinethol or Imuran should be made on the basis of therapeutic response and any toxic effects.

Usage in Pregnancy and Women of Childbearing Age: Zyloprim® (allopurinol) should be used in pregnant women or women of childbearing age only if the potential benefits to the patient are weighed against the possible risk to the fetus.

PRECAUTIONS: Some investigators have reported an increase in acute attacks of gout during the early stages of allopurinol administration, even when normal or subnormal serum uric acid levels have been attained.

It has been reported that allopurinol prolongs the half-life of the anticoagulant, dicumarol. This interaction should be kept in mind when allopurinol is given to patients already on anticoagulant therapy, and the coagulation time should be reassessed.

A fluid intake sufficient to yield a daily urinary output of at least 2 liters and the maintenance of a neutral or, preferably, slightly alkaline urine are desirable to (1) avoid the theoretic possibility of formation of xanthine calculi under the influence of Zyloprim therapy and (2) help prevent renal precipitation of urates in patients receiving concomitant uricosuric agents.

Patients with impaired renal function require less drug and should be carefully observed during the early stages of Zyloprim administration and the drug withdrawn if increased abnormalities in renal function appear.

In patients with severely impaired renal function, or decreased urate clearance, the half-life of oxipurinol in the plasma is greatly prolonged. Therefore, a dose of 100 mg per day or 300 mg twice a week, or perhaps less, may be sufficient to maintain adequate xanthine oxidase inhibition to reduce serum urate levels. Such patients should be treated with the lowest effective dose, in order to minimize side effects.

Mild reticulocytosis has appeared in some patients.

As with all new agents, periodic determination of liver and kidney function and complete blood counts should be performed especially during the first few months of therapy.

ADVERSE REACTIONS:

Dermatologic: Because in some instances skin rash has been followed by severe hypersensitivity reactions, it is recommended that therapy be discontinued at the first sign of rash or other adverse reaction (see WARNINGS). Skin rash, usually maculopapular, is the adverse reaction most commonly reported.
Exfoliative, urticarial and purpuric lesions, Stevens-Johnson syndrome (erythema multiforme) and toxic epidermal necrolysis have also been reported.
A few cases of alopecia with and without accompanying dermatitis have been reported.
In some patients with a rash, restarting Zyloprim (allopurinol) therapy at lower doses has been accomplished without untoward incident.

Gastrointestinal: Nausea, vomiting, diarrhea, and intermittent abdominal pain have been reported.

Vascular: There have been rare instances of a generalized hypersensitivity vasculitis or necrotizing angiitis which have led to irreversible hepatotoxicity and death.

Hematopoietic: Agranulocytosis, anemia, aplastic anemia, bone marrow depression, leukopenia, pancytopenia and thrombocytopenia have been reported in patients, most of whom received concomitant drugs with potential for causing these reactions. Zyloprim® (allopurinol) has been neither implicated nor excluded as a cause of these reactions.

Neurologic: There have been a few reports of peripheral neuritis occurring while patients were taking Zyloprim. Drowsiness has also been reported in a few patients.

Ophthalmic: There have been a few reports of cataracts found in patients receiving Zyloprim. It is not known if the cataracts predated the Zyloprim therapy. "Toxic" cataracts were reported in one patient who also received an anti-inflammatory agent; again, the time of onset is unknown. In a group of patients followed by Gutman and Yü for up to five years on Zyloprim therapy, no evidence of ophthalmologic effect attributable to Zyloprim was reported.

Drug Idiosyncrasy: Symptoms suggestive of drug idiosyncrasy have been reported in a few patients. This was characterized by fever, chills, leukopenia or leukocytosis, eosinophilia, arthralgias, skin rash, pruritus, nausea and vomiting.

OVERDOSAGE: Massive overdosing, or acute poisoning, by Zyloprim has not been reported.

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Cerebrovascular Disease Conference
Mondays, 5-6 p.m., weekly, Rm. 5505. Contact: Wesly Moore, M.D.,Depts. of Surgery, Nurology & Radiology.

Dean's Clinical Rounds
Contact: Louis J. Kettel, M.D.

Dermatology Rounds
Weekly Wed., 11:00 A.M., Contact: Dr. Lynch

Ear, Nose & Throat
Alternate Wed., 4:00 P.M., Contact: Dr. Coulthard

Educaton Bloc
Weekly Thurs, 4:00-5:00 P.M., Rm 5403

Endocrinology Seminar
Weekly Thursday, 12:00-1:00 P.M., Contact: Dr. Nugent

Gastrointestinal Path. Conference
4th Friday, 1:30 P.M.

Head & Neck Tumor Meeting
Alternate Wed., 4:00 P.M., Contact: Dr. Manning

Hematology-Oncology Clinical Conference
Every Tuesday, Noon-1 p.m., 1st, 3rd & 5th Tuesdays, Rm 6505, Tucson Medical Center. 2nd & 4th Tuesdays, Rm. N318, VA Hospital. Contact: Sidney Salmon, M.D., Dept. of Internal Medicine.

Joint Thyroid Clinic Conference
1st & 3rd Mondays of every month. 1 to 2 p.m., AHSC,Rm. 6505. Contact: Sidney Werner, M.D., Dept. of Internal Medicine, Endocrinology Section.

Nuclear Medicine
Thursday, 7:30 a.m., Radiology Conf. Rm., UAHSC. Contact: Irwin M. Freundlich, M.D., Dept. of Diagnostic Radiology.

Neurology Grand Rounds
Every Friday, Noon, 1st, 3rd, & 5th Fridays, UAHSC, Rm. 7505. 2nd & 4th Fridays, VA Hospital. Contact: William A. Sibley, M.D., Dept. of Neurology.

Neuromuscular Disese Conference
Weekly Friday, 11:00 A.M., Contact: Dr. Stern

Neuropathology Review Conference
Thursdays, 8:30 a.m., Rm. 5120, UAHSC. Contact: Peter C. Johnson, M.D.

Neuroradiology Conference
Weekly Thurs., 5:00 P.M., Contact: Dr. P.C. Christenson

Neurosciences Seminar
Every Friday, 7:30 a.m.,UAHSC, Rm 7505. Contact: Colin R. Bamford, M.D., or Jose F. Laguna, M.D., Dept. of Neurology.

Neuromus Journal Conference
2nd & 4th Thursday, 7:00-9:00 P.M., Contact: Dr. Stern.

Orthopedic Rounds
Saturday, 8:00 A.M., Contact: Dr. Peltier.

Pathology Seminar
Friday, 4:30-5:30 p.m., Rm. 5120, UAHSC. Contact: Paul Finley, M.D., Dept. of Pathology.

Pediatric Problem Patient Conference
Weekly Wed., 8:00 A.M., Contact Dr. Walson

Pediatric Research Forum
Weekly Tuesday, 7:30 A.M., Contact: Dr. James Jones

Pediatric Specialty Conference
Weekly Friday, 8:00 A.M., Contact: Dr. Philip Walson.

Psychiatric Grand Rounds
Weekly Wed., 12:00 Noon, Contact: Dr. Robert Cutts

(Psych.) Monthly Case Conference
2nd Friday, 7:30-9:00 A.M., Contact: Dr. Alan Levens

Pulmonary Rounds
Weekly Friday, 11:30 A.M., Contact Dr. Benjamin Burrowns.

Chest Radiology
Monday, 5-6 p.m., weekly, Rm 1535F, UAHSC. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Gastrointestinal Radiology
Mondays, 7:30 a.m., weekly except for summer months. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Radiation Oncology Planning Conference
Friday, weekly, 8:30-10 a.m., UAHSC, Rm. 0655. Contact: Robert S. Heusinkveld, M.D., Radiation Oncology Section.

Radiology-Rheumatology Conference
Thursday, 8 a.m., UAHSC, Library Rm.1535C. Contact: Irwin M. Freundlich, M.D. Dept. of Radiology.

Renal Pathology Conference
1st & 3rd Thurs., 11:30 A.M., Contact: Dr. Nagle.

Resident's Conference
Wednesday, weekly 5-6 p.m., Diag. Radiology Conf. Rm. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology

Uroradiology
Alternate Thurs, 5:00 P.M., Contact I.M. Freundlich

Surgical Literature/Basic Skills
Saturday, 8:00 A.M., Contact: Dr. P. Whitney

Surgical Morbidity & Mortality Conference
Weekly Wed., 8:00 A.M., Contact: Dr. Wangensteen.

Trauma Conference
Every Thursday,4-5 p.m. UAHSC, Rm. 5505. Contact: Martin Silverstein, M.D., Dept. of Surgery.

Grand Ward Rounds—Trauma
Every Tuesday, 8 a.m., UAHSC. Contact: Martin Silverstein, Surgery Dept., Trauma

Toxicology Conference
Weekly Tuesday, 8:00 A.M., Contact: Dr. Keith Likes
Section.

Monthly Meeting Tucson Ultrasonography Group
First Wed. of month, 4:30-5:30 p.m., UAHSC. Contact: Irwin M. Freundlich, M.D.

General Urology Conference
Weekly Thursday, 12:00 Noon, Contact: Dr. G. W. Drach.

PHOENIX VETERANS ADMINISTRATION HOSPITAL

7th St. and Indian School Rd., Phoenix, AZ 85012. Sponsor: VA Hospital. Contact: Alfred Heilbrumn, M.D. Approved for Category 1 credit.

Cancer Symposium
Weekly Wed., 1:00 P.M., Contact Dr. Berk
Ext. 419

Cardiology Conference
2nd Thursday — 1 p.m. — T5,
Habibzadeh, Ext. 348.

Clinicopathology Conference
4th Thursday, March, June, Sept. & Dec., 1
P.M., Contact Dr. Likos, Ext. 623

Dermatology Conference
1st, 2nd, & 3rd Wednesday (usually, but
subject to change) 10 a.m. — C118,
Roberts, Ext. 426.

G.I. Clinic
2nd & 4th Wed, 1:00-4:00 P.M.,

G.I. Radiology Clinical Correlation Conference 1st & 3rd Monday -
p.m. — C435, Dr. Sanowski, Ext. 618.

G.I. Grand Rounds
Weekly, Wed., 8:00 A.M., G-I Ward , Conta
Dr. Severino

G.I. Resident Conference
Weekly Friday, 1:00 P.M., D446, Contac
Drs. Sanowski & Kozarek

G.I. Medical Surgical Conference
Weekly Friday, 2:00 P.M., Path. Conf. Rm.

G.I. Rounds
Weekly Thursday, 1:00 P.M., D446

Medical Service Grand Rounds
Weekly Friday — 11 a.m. — T5, Dr. Zeller, E
272

Medical-Surgical G.I. Conference
1st & 3rd Friday, 2:00 P.M., Rm. 212
Contact: Drs. Kozarek and Mertz, Ext 493

Medical-Surgical Chest Conference
1st and 3rd Thursday — 1 p.m. — T5, D
Chowdhury, Ext. 388.

Surgery-Pathology Conference
Weekly Thursday, 2 p.m. — 2128, Drs. Mert
Ext. 493 & Likos, Ext 623

Surgery Grand Rounds
Weekly Thursday, 3:00 P.M., Contact: D
Mertz, Ext. 493.

TUCSON VETERANS ADMINISTRATION HOSPITAL

3601 South Sixty Ave., Tucson, AZ 85723
Contacts listed below. Aprroved for categor
1 credit.

Medical Surgical Chest Conference
Weekly Tuesday, 2:00 P.M., Contact: Dr. Young

Medical Grand Rounds
Weekly Wed., 12:00-1:00 P.M., V.A. Hospital Staff Conf. Rm. & (AHSC), Contact: Jay Smith, M.D.

Surgical Grand Rounds
Weekly Wed., 4:00 P.M., Contact: Dr. Putnam

Endocrinology Seminar
1st.,3rd, & 5th Thurdsays, 12:00-1:00 P.M., Rm. N318, Contact: Dept. of Internal Medicine.

Vascular Radiology, Interesting Case Conf.
Weekly Thursday, 12:00 Noon

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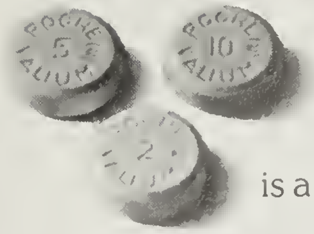

A character all its own.

Valium (diazepam/Roche) is a benzodiazepine with a character all its own. Pharmacologically, it is a potent skeletal muscle relaxant and anticonvulsant (in adjunctive use), as well as an antianxiety agent. Pharmacokinetically, only Valium provides active *diazepam* as well as the active metabolites 3-hydroxydiazepam, desmethyldiazepam and oxazepam.

But the individual character of Valium is even more apparent clinically than pharmacokinetically. And far more significant. That's because of the patient response obtained with Valium. A response which brings a calmer frame of mind. A response which has a pronounced effect on the somatic symptoms of anxiety, particularly muscular tension. A response which helps the patient feel more like himself again because of the way Valium reduces the overwhelming symptoms of anxiety and psychic tension.

Another important aspect of the clinical character of Valium is safety. Though drowsiness, ataxia and fatigue are possible, these and more serious side effects are rarely a problem. Of course, as with all CNS-acting drugs, patients taking Valium should be cautioned against driving, operating dangerous machinery or the simultaneous ingestion of alcohol.

Unquestionably, many psychotherapeutic agents, including other benzodiazepines, have antianxiety effects. But one fact remains: you get a certain kind of patient response with Valium. It's a response you want. A response you know. A response you trust as part of your overall management of anxiety and psychic tension.

Valium® ⓘⓥ
diazepam/Roche
2-mg, 5-mg, 10-mg scored tablets
a prudent choice in psychic tension and anxiety

Before prescribing, please consult complete product information, a summary of which follows:
Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).
The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.
Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.
Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.
Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.
Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).
Supplied: Valium® (diazepam) Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10.

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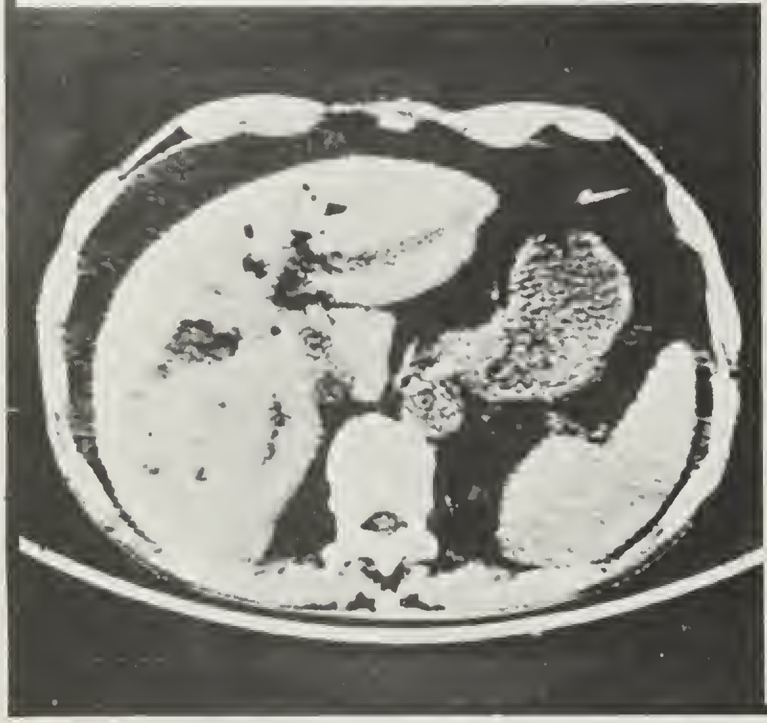

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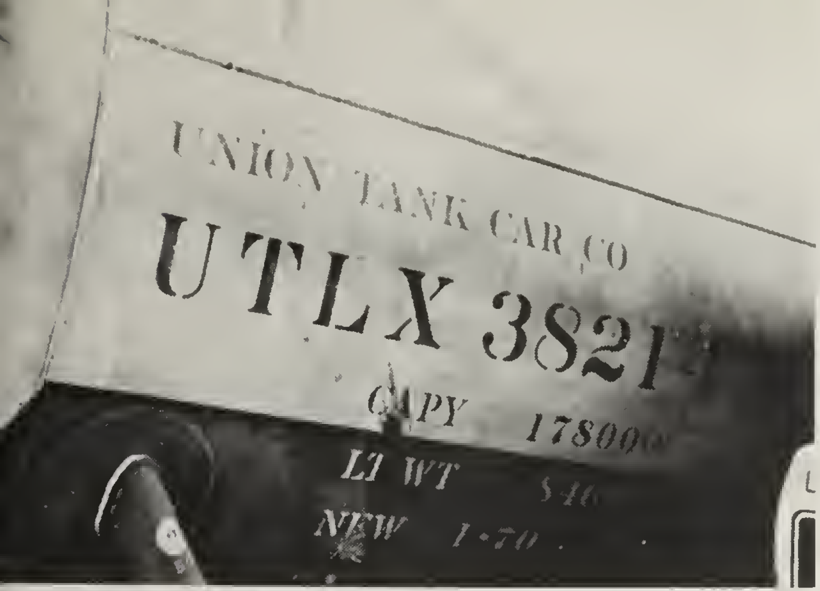

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It was a hot, sunny afternoon in July. On a railroad siding in a small town in Northern Arizona an unforgettable disaster was brewing.

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Again and again the air ambulances returned to the scene, bringing badly-burned patients to Phoenix emergency rooms. Time — the crucial element in this life-saving evacuation. A crisis situation was brought under control.

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an important
in the treatment of

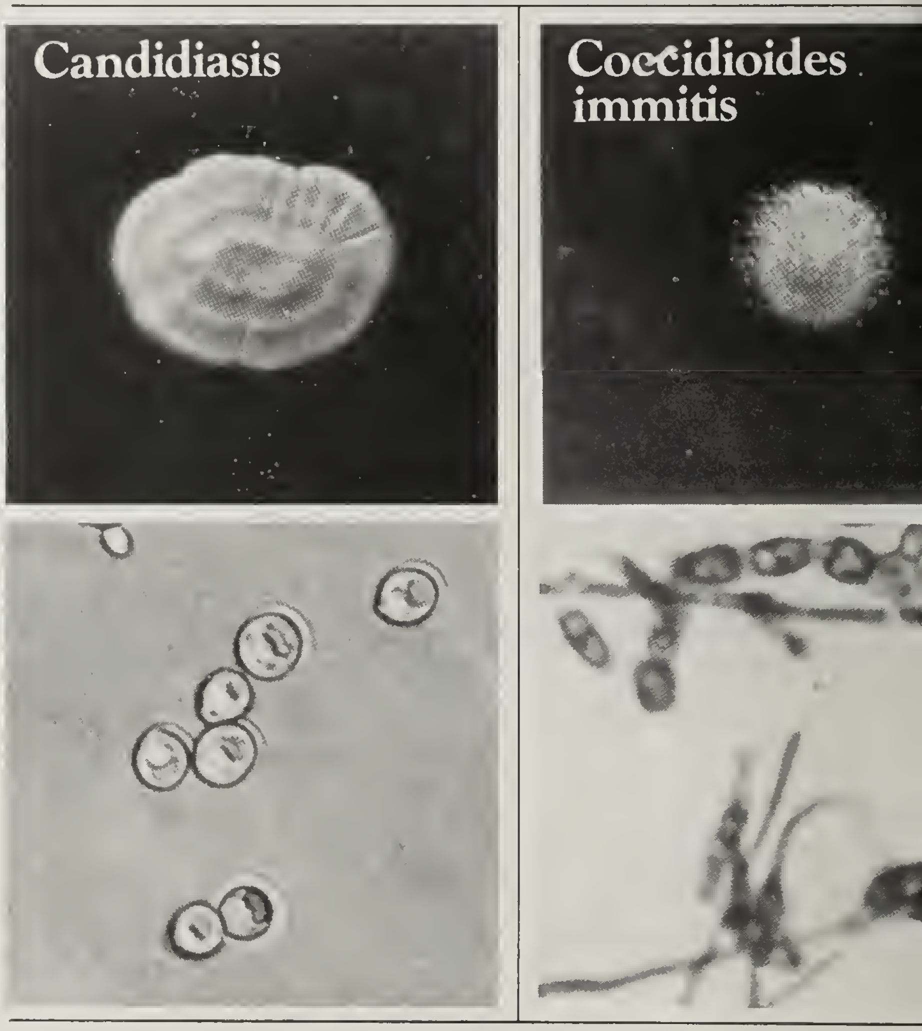
Efficacy of MONISTAT i.v. therapy...

highly favorable rates of recovery or clinical improvement

- effective against systemic candidiasis
  chronic mucocutaneous candidiasis
  coccidioidomycosis
  cryptococcosis
  paracoccidioidomycosis
- in patients who did not respond to amphotericin B therapy
- in immunologically-compromised patients with severe infections

rapid onset of clinical improvement
In most patients who responded, improvement was observed within 1-3 weeks of MONISTAT i.v. therapy; however, prolonged treatment courses may be necessary to prevent relapse.

no development of fungal resistance during therapy reported
In vitro resistance of clinical isolates has not been observed during clinical trials.



Photos courtesy of Dubos, J. R., Bacterial and Mycotic Infections of Man, J. B. Lippincott Company, 1968 and Burrows, W., Textbook of Microbiology, W. B. Saunders Company, 19th editio

and the side effect profile

no nephrotoxicity or other permanent adverse effects in clinical studies

- even in prolonged therapy
- even in patients with renal disease

extensively evaluated
During clinical trials in over 400 patients, no permanent adverse reactions were reported.

Adverse reactions
MONISTAT i.v. should not be administered undiluted. Rapid injection of the undiluted solution may produce transient tachycardia or arrhythmia.

Adverse reactions include phlebitis, pruritus, rash, nausea, vomiting, febrile reactions, drowsiness, diarrhea, anorexia and flushes. Transient hematologic changes have occurred; therefore, the patient should be closely monitored.

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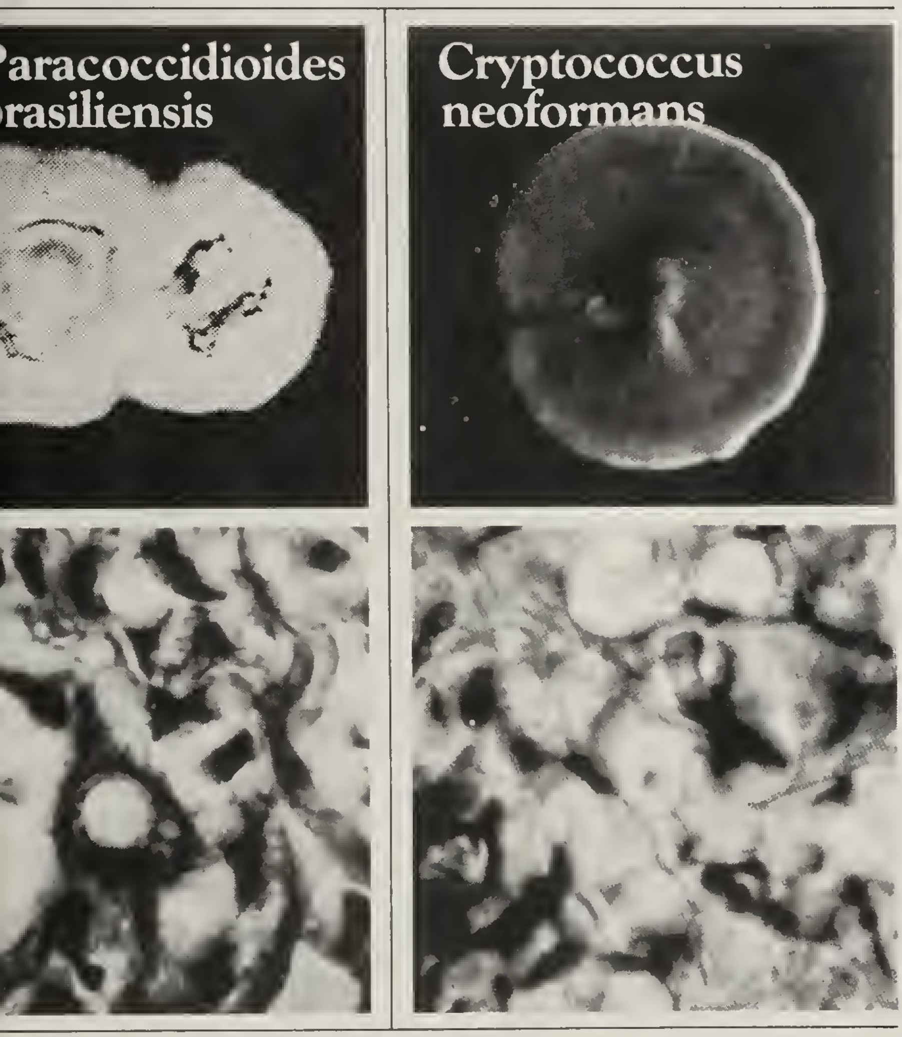
advancement
systemic mycoses



generally well tolerated by severely debilitated patients

- cancer patients
- patients receiving immunosuppressive drugs
- patients with serious underlying conditions such as renal insufficiency or diabetes mellitus

for more information

Because of the uniqueness of MONISTAT i.v. only some of the highlights can be presented in this advertisement. For more information, i.e., product monograph, dosage schedules, etc., please fill out the coupon on the next page and mail to Ortho Pharmaceutical Corporation.

Please turn page for Prescribing Information

Ortho Pharmaceutical Corporation
Raritan, New Jersey 08869

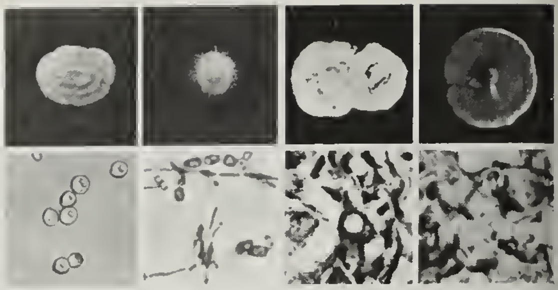
Monistat i.v.

TRADEMARK

(miconazole)

broad-spectrum activity against major systemic fungal pathogens

systemic fungal infections…



Monistat i.v.

TRADEMARK

(miconazole)

a broad-spectrum systemic antifungal agent

DESCRIPTION: MONISTAT i.v. (miconazole) 1-{2-(2,4-dichlorophenyl)-2-[(2,4-dichlorophenyl) methoxyl ethyl} 1 H-imidazole is a synthetic antifungal supplied as a sterile solution for intravenous infusion. Each ml of this solution contains 10 mg of miconazole with 0.115 ml PEG 40 castor oil, 1.0 mg lactic acid USP, 0.5 mg methylparaben USP, 0.05 mg propylparaben USP in water for injection. Miconazole i.v. is a clear, colorless to slightly yellow solution having a pH of 3.7 to 5.7.

CLINICAL PHARMACOLOGY: MONISTAT i.v. is rapidly metabolized in the liver and about 14% to 22% of the administered dose is excreted in the urine, mainly as inactive metabolites. The pharmacokinetic profile fits a three-compartment open model with the following biologic half-life: 0.4, 2.1, and 24.1 hours for each phase respectively The pharmacokinetic profile of MONISTAT i.v is unaltered in patients with renal insufficiency, including those patients on hemodialysis. The in/vitro antifungal activity of MONISTAT i.v. is very broad Clinical efficacy has been demonstrated in patients with the following species of fungi. *Coccidioides immitis, Candida albicans, Cryptococcus neoformans, and Paracoccidioides brasiliensis.*

Recommended doses of MONISTAT i.v. produce serum concentrations of drug which exceed the in-vitro MIC values for the fungal species noted above. Doses above 9 mg/kg of MONISTAT i.v. produce peak blood levels above 1μg/ml in most cases. The drug penetrates into joints.

INDICATIONS: MONISTAT i.v. is indicated for the treatment of the following severe systemic fungal infections: coccidioidomycosis, candidiasis, cryptococcosis, paracoccidioidomycosis, and for the treatment of chronic mucocutaneous candidiasis. However, in the treatment of fungal meningitis and urinary bladder infections an intravenous infusion alone is inadequate. It must be supplemented with intrathecal administration and bladder irrigation. Appropriate diagnostic procedures should be followed and MICs should be determined.

MONISTAT i.v. should not be used to treat common trivial forms of fungal diseases.

CONTRAINDICATIONS: MONISTAT i.v. is contraindicated in those patients who have shown hypersensitivity to it.

WARNINGS: Rapid injection of undiluted MONISTAT i.v., may produce transient tachycardia or arrhythmia.

PRECAUTIONS: Before a treatment course of MONISTAT i.v is started, the physician should make sure that the patient is not hypersensitive to the drug product. MONISTAT i.v should be given by intravenous infusion. The treatment should be started under stringent conditions of hospitalization but subsequently may be given to suitable patients under ambulatory conditions with close clinical monitoring It is recommended that an initial dose of 200 mg be given with the physician in attendance. It is also recommended that clinical laboratory monitoring including hemoglobin, hematocrit, electrolytes and lipids be performed.

It should be borne in mind that systemic fungal mycoses may be complications of chronic underlying conditions which in themselves may require appropriate measures.

Pregnancy: Reproductive studies with MONISTAT i.v. in rats and rabbits revealed no evidence of impaired fertility or harm to the fetus. There are no data, however, on the use of the drug in pregnant women.

Children: Since the safety of miconazole i.v. in children under one year of age has not been extensively studied, its benefits in this age group must be weighed against the possible risks involved

ADVERSE REACTIONS: Adverse reactions which have been observed with MONISTAT i.v therapy include phlebitis, pruritus, rash, nausea, vomiting, febrile reactions, drowsiness, diarrhea, anorexia and flushes. In the U.S. studies, 29% of 209 patients studied had phlebitis, 21% pruritus, 18% nausea, 10% fever and chills, 9% rash, and 7% emesis. Transient decreases in hematocrit and serum sodium values have been observed following infusion of MONISTAT i.v. Thrombocytopenia has also been reported. No serious renal or hepatic toxicity has been reported If pruritus and skin rashes are severe, discontinuation of treatment may be necessary. Nausea and vomiting can be mitigated with antihistaminic or antiemetic drugs given prior to MONISTAT i.v. infusion, or by reducing the dose, slowing the rate of infusion, or avoiding administration at mealtime.

Aggregation of erythrocytes or rouleau formation on blood smears has been reported Hyperlipemia has occurred in patients and is reported to be due to the vehicle. Cremophor EL (PEG 40 castor oil).

DRUG INTERACTIONS: Drugs containing cremophor-type vehicles are known to cause electrophoretic abnormalities of the lipoprotein. These effects are reversible upon discontinuation of treatment but are usually not an indication that treatment should be discontinued

Interaction with the coumarin drugs resulting in an enhancement of the anticoagulant effect has also been reported In cases of simultaneous treatment with MONISTAT i.v. and coumarin drugs, the anticoagulant effect should be carefully titrated since reductions of the anticoagulant doses may be indicated.

DOSAGE AND ADMINISTRATION

DOSAGE

Adults. The doses may vary with the diagnosis and with the infective agent, from 200 to 1200 mg per infusion. The following daily doses, which may be divided over 3 infusions, are recommended:

Organism	Dosage Range*	Duration of Successful Therapy (weeks)
Coccidioidomycosis:	1800 to 3600 mg per day	3 to > 20
Cryptococcosis:	1200 to 2400 mg per day	3 to > 12
Candidiasis:	600 to 1800 mg per day	1 to > 20
Paracoccidioidomycosis:	200 to 1200 mg per day	2 to > 16

*May be divided over 3 infusions.

Repeated courses may be necessitated by relapse or reinfection.

Children: A total daily dose of about 20 to 40 mg/kg is generally adequate. However, a dose of 15 mg/kg body weight per infusion should not be exceeded.

ADMINISTRATION: MONISTAT i.v. should be diluted in at least 200 ml of fluid. The diluent of choice is 0.9% sodium chloride or alternatively Dextrose 5% injectable solution. The intravenous infusion should be given over a period of 30 to 60 minutes.

Generally, treatment should be continued until all clinical and laboratory tests no longer indicate that active fungal infection is present. Inadequate periods of treatment may yield poor response and lead to early recurrence of clinical symptoms. The dosing intervals and sites and the duration of treatment vary from patient to patient and depend on the causative organism.

OTHER MODES OF ADMINISTRATION: Intrathecal. Administration of the undiluted injectable solution of MONISTAT i.v. by the various intrathecal routes (20 mg per dose) is indicated as an adjunct to intravenous treatment in fungal meningitis. Succeeding intrathecal injections may be alternated between lumbar, cervical, and cisternal punctures every 3 to 7 days. Bladder instillation of 200 mg of diluted solution is indicated in the treatment of mycoses of the urinary bladder.

HOW SUPPLIED: MONISTAT i.v. is supplied in 20 ml ampoules.

MONISTAT i.v. (miconazole) is an original product of Janssen Pharmaceutica N.V., Belgium, and co-developed by Ortho Pharmaceutical Corporation.

Ortho Pharmaceutical Corporation
Raritan, New Jersey 08869

Ortho Pharmaceutical Corporation AM-8-79
c/o 733 Summer Street
Stamford, CT 06901

Please send me more information on the broad-spectrum, systemic antifungal agent... MONISTAT i.v. (miconazole).

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Specialty____________________

October 1978

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Tenuate® CIV
(diethylpropion hydrochloride NF)

Tenuate Dospan®
(diethylpropion hydrochloride NF) controlled-release

AVAILABLE ONLY ON PRESCRIPTION
Brief Summary
INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below
CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma. Agitated states Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).
WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle; the patient should therefore be cautioned accordingly *Drug Dependence*: Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended. Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy*: Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children*: Tenuate is not recommended for use in children under 12 years of age.
PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary.
ADVERSE REACTIONS: *Cardiovascular*: Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System*: Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal*: Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic*: Urticaria, rash, ecchymosis, erythema. *Endocrine*: Impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System*: Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous*: A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.
DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in midevening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg. tablet daily, swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.
OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of April, 1976

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Direct Medical Inquiries to
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References: 1. Citations available on request — Medical Research Department, MERRELL RESEARCH CENTER, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215. 2 Hoekenga, M.T., O'Dillon, R.H., and Leyland, H.M. A Comprehensive Review of Diethylpropion Hydrochloride. International Symposium on Central Mechanisms of Anorectic Drugs, Florence, Italy, Jan. 20-21, 1977.

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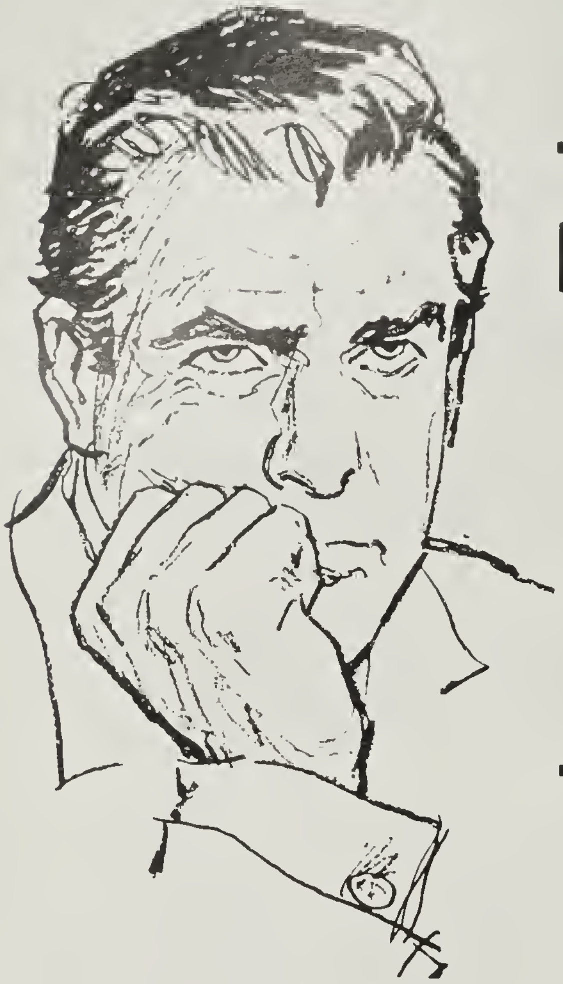

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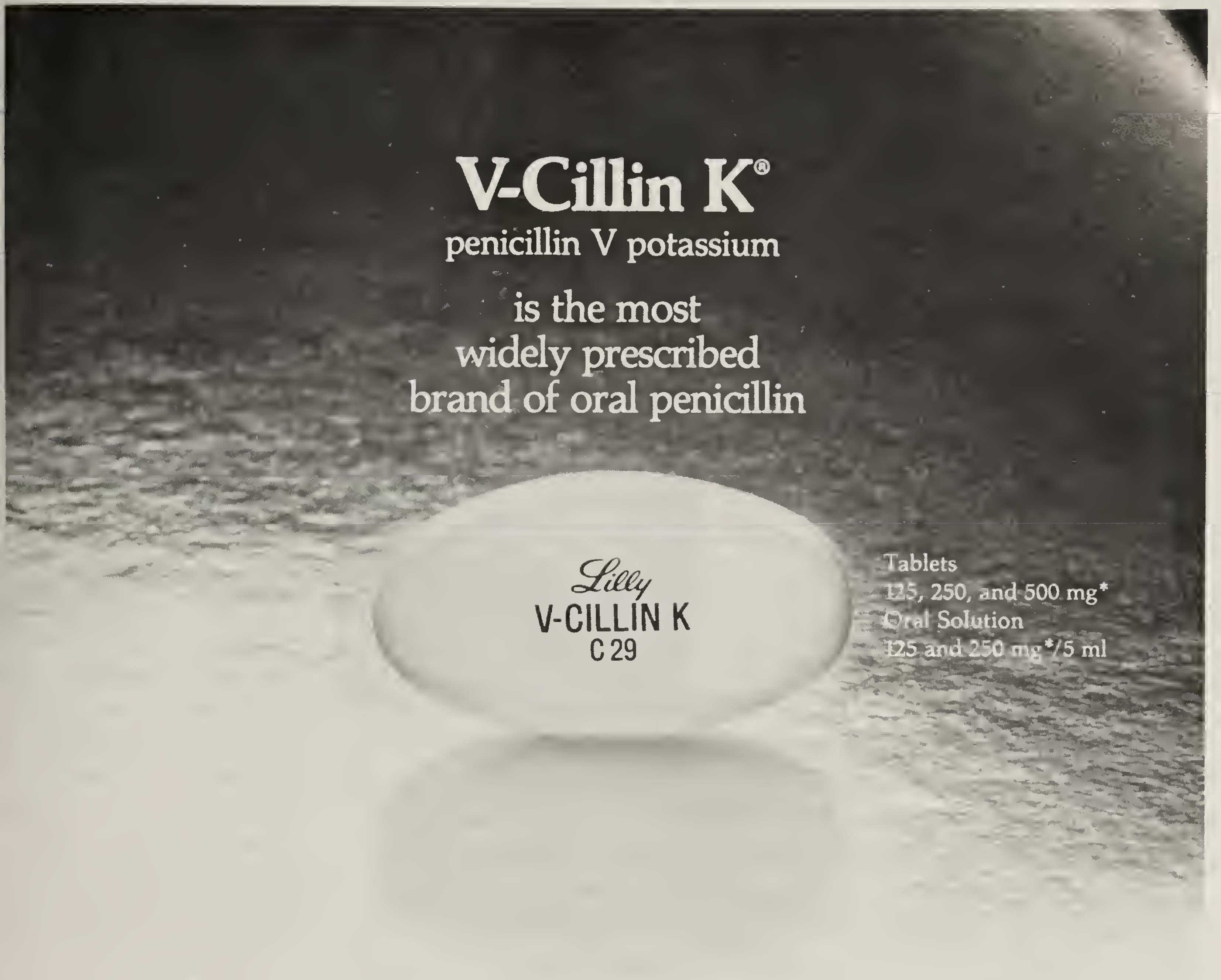

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Warnings: Serious, occasionally fatal, anaphylactoid reactions have been reported. Some patients with penicillin hypersensitivity have had severe reactions to a cephalosporin; inquire about penicillin, cephalosporin, or other allergies before treatment. If an allergic reaction occurs, discontinue the drug and treat with the usual agents (e.g., epinephrine or other pressor amines, antihistamines, or corticosteroids).

Precautions: Use with caution in individuals with histories of significant allergies and/or asthma. Do not rely on oral administration in patients with severe illness, nausea, vomiting, gastric dilatation, cardiospasm, or intestinal hypermotility. Occasional patients will not absorb therapeutic amounts given orally. In streptococcal infections, treat until the organism is eliminated (minimum of ten days). With prolonged use, nonsusceptible organisms, including fungi, may overgrow; treat superinfection appropriately.

Adverse Reactions: Hypersensitivity, including fatal anaphylaxis. Nausea, vomiting, epigastric distress, diarrhea, and black, hairy tongue. Skin eruptions, urticaria, reactions resembling serum sickness (including chills, edema, arthralgia, prostration), laryngeal edema, fever, and eosinophilia. Infrequent hemolytic anemia, leukopenia, thrombocytopenia, neuropathy, and nephropathy, usually with high doses of parenteral penicillin. (102175)

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The Maker

Examining a Few Myths About Prescribing.

Increasing pressure is being put on the practicing physician to prescribe drugs generically. You are told that brand–name products are universally “expensive” and generic versions are relatively “cheap.” To make this case, the most extreme (rather than typical) price differentials are cited. Thus, consumers are led to believe that such differentials are commonplace. Even your knowledge and your motives as a physician are questioned.

Understandably, these views have created myths. We think it’s time to examine them in the light of all the facts and ramifications.

MYTH: There are no differences in quality and performance between brand-name products and their generic counterparts. The corollary is that there are no differences among products made by high-technology, quality-conscious, research-based companies and those made by commodity-type suppliers.

FACT: The Food and Drug Administration does a good job in monitoring a generally excellent drug supply. Still, it has nowhere near the resources to guarantee the quality and bioavailability of all marketed products at any given time. Just a few months ago, for example, it noted that batches of tetracycline HCl capsules which met official monograph requirements were not bioequivalent to a reference product. As y know, there is substanti literature on this subjec affecting many drugs, i cluding such antibiotics as tetracycline and erythromycin. The record drug recalls and court actions affirms strongly that there are difference among pharmaceutical companies and their products. Research-intensive companies have far better records than those that do no research and may practice minimum quality assurance.

MYTH: Industry favors only “expensive” brand names and denigrates all generics.

FACT: PMA companies make 90 to 95 percent o the drug supply, including, therefore, most of th generics. Drug nomenclature is not the important point; it’s the competence of the manufacturer and the integrity o the product that count.

Matters.

IYTH: Generic options al-
ost always exist.

ACT: About 55 percent
f prescription drug ex-
enditure is for single-
ource drugs. This
eans, of course, that for
nly 45 percent of such
xpenditure, is a generic
rescribing option avail-
ble.

IYTH: Generic
rescriptions are filled with
expensive generics, thus
aving consumers large
ums of money.

ACT: Market data show
hat you invariably
rescribe—and pharma-
ists dispense—both
rand and generically
abeled products from
nown and trusted
ources, in the best inter-
st of patients. In most
ases the patient receives
proven brand product.
avings from voluntary
r mandated generic
rescribing are grossly
xaggerated.

MYTH: Drugs account for a major portion of the rise in health care costs.

FACT: Drugs represent a very small part of such costs. The amount of the health care dollar spent for prescription drugs was about 12 cents in 1967; today it is about 8 cents. And you as a physician are most conscious of how drug therapy can cut hospitalization, avert surgery, reduce office visits and keep patients on the job.

MYTH: Government intrusions into the marketplace will save tax money.

FACT: Government schemes always cost the taxpayer something, and the costs often exceed the benefits. Certainly, any federal "help," such as lists of wholesale drug prices sent to all physicians and pharmacists, will be no exception. Just think of the expense of keeping them current! Moreover, wholesale prices are poor guides to actual transaction prices and even worse guides to retail prices.

The PMA Position

We believe your freedom to prescribe, either by generic or brand name, should be totally unabridged. Otherwise, your prescribing prerogatives and your relationships with patients will be seriously impaired.

The maker does matter

After the myths about price and equivalency have been shattered, one fact stands out more clearly than ever: *The maker does matter.* As always, your best guide to drug therapy for your patients is to select products—both brands and generics—from manufacturers with credentials and performance records you have come to respect.

PMA

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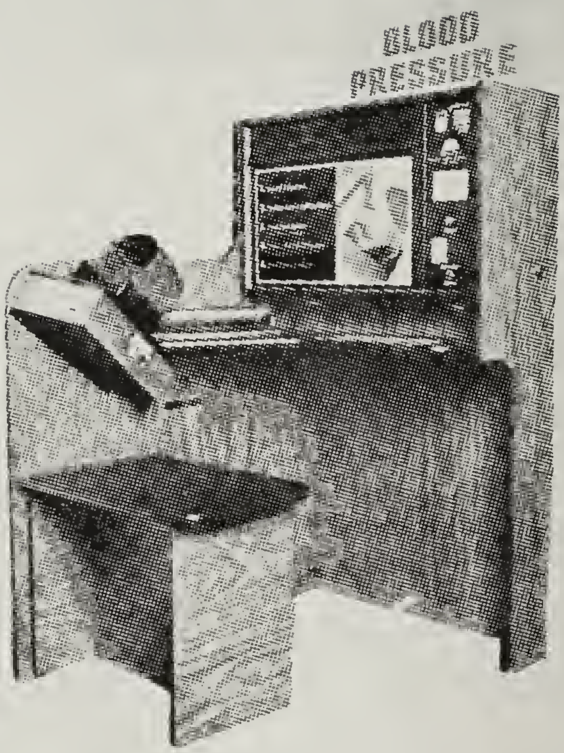


SOME RESEARCH RESULTS

Dr. David M. Berkson re: study on Vita-Stat Computer as conducted by the Chicago Department of Health and reported in the American Journal of Public Health May 1979 states:

> **"Our results indicate that when the machine and a trained observer measure blood pressure simultaneously, there is very good agreement."**

SRI International (formerly Stanford Research Institute)—
report of Joseph H. Chadwick, Ph.D., Director, Health Systems Programs:

> **"The Vita-Stat machine appears to me to be as good as a well-trained blood pressure technician."**

Dr. R. Patterson Russell, Associate Professor of Medicine at Johns Hopkins Medical Institution, Director of the Hypertension Clinic at Johns Hopkins Hospital and Chairman of the High Blood Pressure Commission of Maryland, state the following in the March, 1979 issue of "Preventive Medicine," the official Journal of the American Health Foundation:

> **"Blood pressure measurements by the Vita-Stat correlated as well as did measurements by physicians and provided reliable determinations suitable for the purposes of screening, referral and monitoring of treatment."**

Cardiology

New Direction in Cardiac Valve Replacement

Neal W. Salomon, M.D.

In the eighteen years since the first successful cardiac valve replacement, dramatic advances have been made in heart valve replacement technology. Developments in both applied and basic engineering have lead to improved hydraulic design and greater durability of both mechanical and tissue valves. In spite of the development of biocompatible, durable metallic prostheses with central laminar flow characteristics, thromboembolism and hemorrhagic episodes from anticoagulation have been major causes of morbidity. It has been estimated that thromboembolism occurs at the rate of approximatley 5% per patient year, with a similar incidence of anticoagulation-related hemorrhage. Dissatisfaction with these complications had lead to the increasing use of tissue valves which presently comprise approximately 65% of all cardiac valve replacements during the past year in the United States.

Although prosthetic valves are constructed with normal valvular characteristics in mind, even with present day technology, we have been unable to imitate the flow dynamics and biologic properties of normal valvular tissue. Consequently, mechanical valves used presently, although markedly improved from earlier models, still are characterized by unphysiologic flow characteristics. Because of this, and the obligatory use of non-biologic materials, prosthetic valves have been associated with significant risk of structural failure, infection (approximately 1% per patient year), poppet dusfunction, thrombosis and embolism (the latter two approximately 5% per patient year). In addition, the almost universal need for life-long anticoagulation imposes a considerable risk of hemorrhagic complications with an incidence of approximately 5% per patient year.

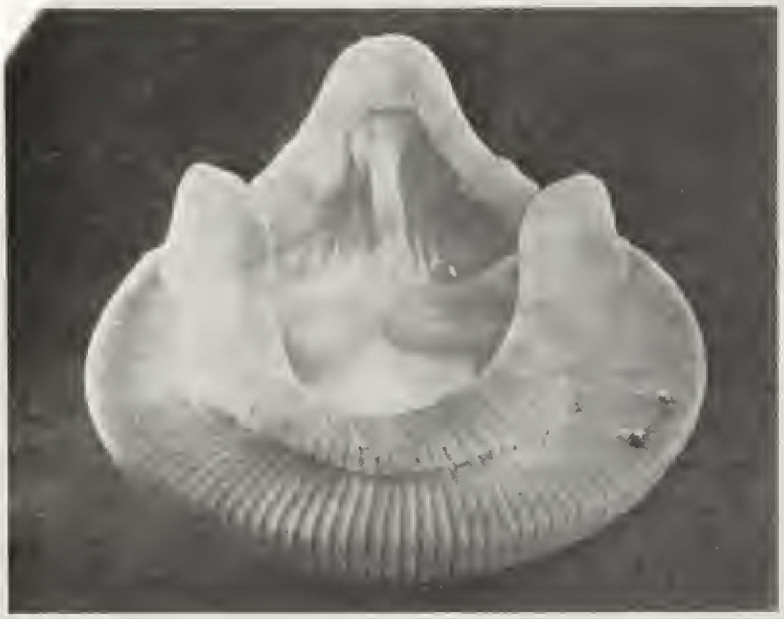
With these consideration in mind, there has been a growing interest in the development of biologic or tissue valves. The major advantages of tissue valves are the simulation of normal flow characteristics and the use of "natural" or "biologic" materials with a lower propensity toward thrombosis and embolization. In the past, the major disadvantage of virtually all tissue valves had been the lack of durability and the progressive degeneration of the valvular tissue. Aortic homografts recovered from human cadavers were used in the mid-1960's. They were found to develop regurgitation in about 50% of cases but the incidence of thromboembolism was nearly zero, and although a high percentage of patients underwent a second valve replacement to correct the insufficiency, the overall survival was comparable to those patients with mechanical prostheses. Developments during the past six or seven years have altered this picture, and in fact, have significantly reversed previous trends in the selection of heart valve substitutes.

Tissue valves have been made from autologous tissue harvested from the patient (fascia lata, pulmonary valve, pericardium), homologous tissue from cavaders (aortic valves), or heterologous tissue from other animal species (procine aortic valve, bovine pericardium). The most promising of the tissue valves to date is the porcine heterograft aortic valve which has virtually revolutionized the valve replacement market.

The porcine aortic heterograft has been in wide-spread clinical use for approximately 8 years. The valves are prepared in a buffered glutaraldehyde solution which preserves the delicate cusp architecture and pliability while converting the valve into a non-viable biologically inert structure. This method of preservation has resulted in a valve which is functionally durable, minimally thrombogenic and hemodynamically acceptable, except in the smallest sizes, in all valve positions for the present follow-up duration of 6-8 years.

As clinical experience with the porcine aortic heterograft grows, it has become apparent that this valve has the lowest incidence of prosthesis-related complications of any valve substitute yet developed [1, 2].

From: Univ. of Arizona Health Sciences Center, Sect. of Cardiovascular and Thoracic Surgery, Tucson, AZ 85724.



The incidence of mechanical complications such as thrombosis, calcification and perforation is extremely low and for both aortic and mitral valves the incidence of prosthesis-related mortality during a 6-8 year period is essentially zero.[3,4] The reoperation rate for replacement of failed heterograft valves is the lowest of any valve substitute and approximates 0.5% over a seven year period. The overall incidence of thromboembolism has also remained very low,[5] approximating that for normal native valves. Over a five year period of study for both mitral and aortic valves, the approximate rate of thromboembolic events per patient year was less than 0.5% This is despite the fact that virtually none of the patients with aortic valve replacement and only some of the patients with mitral valve replacement (only those in chronic atrial fibrillation or with a history of previous thromboembolism) with heterograft valves were maintained on chronic coumadin anticoagulation. The infection rate on the heterograft valves is approximately 1% per patient year which is the same as that found with mechanical prostheses.[3]

As can be seen from the figure, the porcine heterograft valve closely resembles a normal human aortic valve and is mounted in such a way as to be suitable for replacement of any diseased cardiac valve. The major advantages of this valve are the central unobstructed orifice and the consequently normal flow characteristics and lack of turbulence in contrast to the prosthetic mechanical valves. In order for the valve leaflets to have sufficient support, however, rather bulky supporting posts or stents, have been included in the valve design. Because of this and because there is only partial opening of one of the valve leaflets, there is a relatively smaller effective valve orifice as compared to either a normal human valve or to several of the current models of prosthetic valves. This consideration is only significant when using the smaller valve sizes (23mm diameter or less) but may be important enough to preclude the use of this type of valve when such a small size is necessary. Further structural improvements are being made and should provide a more effective orifice to valve annulus ratio.

Porcine heterograft valves are currently made by several manufacturers in the United States and abroad with Hancock Laboratories, Anaheim, California producing approximately 90% of all tissue valves. Shiley Laboratories manufactures the Angell-Shiley porcine heterograft which is comparable to the Hancock valve. More recently the Ionescu-Shiley gluteraldehyde-stablized bovine pericardial heterograft valve has gained acceptance as a durable and even more hemodynamically favorable heart valve substitute.[6] Because of the excellent hydraulic performance of this valve, it is currently highly recommended when very small valve sizes are required. The nationwide trend toward the use of tissue valves is evidenced by the fact that approximatley 60-65% of all heart valve substitutes currently implanted are tissue valves. This is a dramatic alteration from earlier years before suitable bioprostheses were available.

At the University of Arizona Health Sciences Center we have used these bioprostheses virtually exclusively with no instances of valve failure or thromboembolism during the past one and one-half years. Patients are followed postoperatively with periodic clinic visits and routine auscultation. No special noninvasive examinations are performed in the absence of clinical problems. We have found conventional M-mode echocardiography to be of limited value in assessing valve function and are currently evaluating a new technique of 2-dimensional echocardiography which allows excellent visualization of any heart valve substitute. From a practical point of view, however, no routine studies are required and if the patient does develop a clinical problem suggestive of valve malfunction, cardiac catheterization and angiography are usually indicated. Tissue valves are currently being implanted at a rate of more that 1,200 per month throughout the world and if their structural integrity is maintained for several more years, this valve will become the standard against which other valve substitutes will be compared.

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Seminars in Chest Medicine

Fiberoptic Bronchoscopy

David H. Drachler, M.D.

Editors:
Faysal M. Hasan, M.D.
Richard I. Lemen, M.D.
Bernard E. Levine, M.D.

Introduction

The flexible fiberoptic bronchoscope was introduced in 1968 by Ikeda and represents the most important and new diagnostic aid in pulmonary medicine since the advent of the chest roentgenogram. Fiberoptic bundles transmit light from an external source and conduct the visual image from the distal lens to the eyepiece. An adjacent channel permits suctioning, bronchial brushings, and bronchial biopsies. The tip is capable of 130 to 180 degrees of angulation and can be guided to the evel of the fourth to sixth order subsegmental bronchi. Its advantages over the rigid bronchoscope include the ncreased view and biopsy range, the ease of administration, and the greater patient acceptance. Furthermore, the flexible scope can be used without nterrupting mechanical ventilation, can be inserted through artificial airways, and is not difficult in patients with head and neck lesions.

Procedure

Fiberoptic bronchoscopy should be performed in a specially equipped endoscopy room. However, the procedure may be undertaken in the emergency room, ntensive care unit, operating room, or at the bedside if necessary. Although the patients are usually hospitalized, flexible fiberoptic bronchoscopy can be accomplished safely in selected outpatients.[1]

The patient is advised not to take anything by mouth for at least four hours prior to the examination. Parenteral premedication is given to provide mild sedation, to reduce bronchorrhea, to minimize bronchospasm, and to block the vasovagal reflex. General anesthesia is never required. Topical anesthesia is delivered to eliminate the gag reflex, to produce vocal cord and endobronchial anesthesia, and to anesthetize the nasal mucosa when the instrument is passed transnasally. The nasal passageway is more popular than the transoral route because of enhanced patient tolerance. However, the transnasal approach does not permit insertion of the endoscope through an endotracheal tube which is recommended by some to provide optimal protection to the airway.[2] While this issue remains controversial, the use of an oral endotracheal tube or the passage of the fiberscope through a rigid bronchoscope is universally advisable when the airway is likely to become compromised by brisk hemorrhage. Since the external diameter of the fiberscope is 5.8-6 mm., the internal diameter of the endotracheal tube must be at least 8.5 mm. to avoid serious airflow obstruction. Special adapters permit endoscopy while the patient remains on continuous mechanical ventilation. When just supplemental oxygen is needed, a nasal oxygen cannula will suffice.

Indications

The most common diagnostic indications for flexible fiberoptic bronchoscopy include hemoptysis, roentgenographic abnormalities, endobronchial lesions, and atelectasis. While massive hemoptysis (greater than 30 cc./24 hours) is more safely evaluated using the enhanced suctioning capabilities and fixed airway of rigid bronchoscopy, the fiberoptic bronchoscope is more helpful to search for the site of mild or moderate bleeding in subsegmental airways.[3]

Fiberoptic bronchoscopy is very useful in the assessment of patients with suspected bronchogenic carcinoma. The diagnostic yield of both the brushings and the transbronchial biopsy depends upon the tumor location, size, and histology, as well as upon the skills of the endoscopist and the cytopathologist. When the tumor is central and directly visible, the diagnostic yield is 86-94%.[4] [5] When a lesion is periperal and therefore not amendable to biopsy or brushing under direct vision, the yield may fall to 30% of cases.[4] With fluoroscopic guidance of the brushes and forceps to the peripheral lesions, positive findings may occur in 80% of cases.[4] The farther from the hilum and the smaller the tumor diameter, the poorer the yield.[6] Histology also affects the diagnostic yield as the exfoliated cells of adenocarcinoma are much more difficult to recognize than those of epidermoid and oat cell carcinoma.[7] The localization of radiographically occult lung cancer is another oncological application of the fiberoptic bronchoscope. However, the clinical usefulness of the fiberoptic bronchoscope in the screening of high risk patients for lung carcinoma is still uncertain.[8]

The immuno-suppressed patient with possible pulmonary infections is another candidate for fiberoptic bronchoscopy. The combination of bronchial brushings, transbronchial biopsies, and bronchial washings is particularly useful in the diagnosis of nonbacterial lung

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From: Affiliated Chest Physicians, Ltd., 555 W. Catalina Drive, Ste 221, Phoenix, AZ 85013

thogens. Pneumocystis carinii, nocardia, lastomycosis, tuberculosis, aspergillosis, histoplasmosis, cytomegalovirus, candidiasis, cryptococcosis, and coccidioidomycosis have all been identified with the fiberoptic bronchoscope.[9-12] Bronchoscopy may, however, be misleading for routine aerobic and anaerobic bacterial infections.[13]

Transbronchial biopsy has also been helpful in the evaluation of diffuse parenchymal lung disease. Sarcoidosis, usual interstitial pneumonitis, radiation pneumonitis, Goodpasture's syndrome, lymphangitic carcinoma, metastatic cancer, granulomatous disease, collagen disease, pneumoconiosis, alveolar cell carcinoma, lymphoma, lipoid pneumonia, and alveolar proteinosis are all among the list of diagnoses obtainable by fiberoptic bronchoscopy.[14, 15]

The inspection of the vocal cords and of the endobronchial tree is another important use of the procedure. Vocal cord lesions and paralysis, tracheal tumors, deviation, inflammation, and stenosis, and central endobronchial lesions can be indentified endoscopically. The instrument also permits the recognition of a fractured bronchus, endotracheal burn, proximal mucus plugging, foreign body apsiration, carinal abnormalities, artificial airway problems, and suspected aspiration.

Although the suctioning channel in the fiberoptic bronchoscope is small (1.5-2.6 mm.), the instrument has several therapeutic roles. Subsegmental lavage for secretion retention can be helpful in the patient who is unable to raise secretions due to coma, diaphragmatic paralysis, chest wall or abdominal pain, severe mucoid impaction, or mechanical ventilation. Beneficial endobronchial lavage has also been demonstrated in cystic fibrosis.[16] Flexible fiberoptic bronchoscopy may also be used to aspirate the contents of a lung abscess. The use of the fiberoptic bronchoscope for the removal of foreign bodies is still controversial. The retrieval of foreign bodies by this instrument has been facilitated by specially designed basket, back-loading grasping, and four-pronged forceps. While the recovery of coins, stones, hardware, buttons, bones, teeth, plastics, etc. have all been documented with the flexible fiberoptic bronchoscope, the extraction of foreign bodies requires particular skill, special equipment, and a protected airway.[17] Another therapeutic function of the fiberoptic bronchoscope is that it can be used to guide an oral endotracheal tube in the patient who is difficult to intubate in the conventional manner.

Complications

Given the widespread use of flexible fiberoptic bronchoscopy in critically ill patients and in training programs, the fact that major complications, requiring special resuscitative measures, occurred in only 0.08% of procedures is extraordinary.[18] Hemorrhage exceeding 50 ml. and 100 ml. has been documented in 1.3% and 2% of procedures respectively, while pneumothoraces have occurred in 4-5.5% of cases.[11, 19] A questionnaire survey of 48,000 examinations listed only 41 "life-threatenin reactions" to anesthesia, and 52 episodes of "life threatening airway complications."[20] Fever, hypoxia transient arrhythmias, vasovagal reaction, pneumonitis hoarseness, bronchospasm, and aspiration are othe risks of endoscopy. Death has been reported in 0.1-0.2% of procedures and has been attributed to hemorrhage tension pneumothorax, and cardiac arrest.[18 1] Underlying coagulation abnormalities, cardiac disease severe hypoxia, hematological malignancies, and age ar recognized risk factors.

Summary

Flexible fiberoptic bronchoscopy has greatly enhanced our diagnostic capabilities. It is preferable to the rigi bronchoscope for the evaluation of endobronchia disease, pulmonary nodules and diffuse lung disease, no only because of its superior visualization but als because of its excellent patient tolerance, low incidenc of complications, and ease of application. While it greatest impact has been in the identification o bronchogenic carcinoma, it has also been helpful i patients with opportunistic lung infection, secretio retention, and interstitial lung disease. The flexibl fiberoptic bronchoscope can be introduced through ora endotracheal and tracheostomy tubes, need no interrupt mechanical ventilation, and can be used to hel guide the insertion of oral endotracheal tube in patient who are otherwise difficult to intubate.

However, for all its usefulness, there are importan reservations. The rigid bronchoscope remains th instrument of choice in cases of massive hemoptysis vascular tumors, and foreign body aspiration Percutaneous needle aspiration biopsy is ofte preferable for obtaining the diagnosis of very periphera lung parenchymal lesions, and open lung biopsy has greater diagnostic yield in diffuse lung disease Furthermore, there are areas of the endobronchial tre which are not accessible due to flexion limitations of th bronchoscope. Nevertheless, this innovativ nonthoracotomy technique adds a new diagnostic an therapeutic dimension with low mobidity for a vas number of patients with pulmonary disease.

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Dean's Clinical Rounds

Clinical Aspects of Pain

Part II

Stephen B. Shanfield, M.D.
Stuart R. Hameroff, M.D.
John T. Boyer, M.D.
Stephen S. Cox, M.D.

Pain in the Arthritic Patient

John T. Boyer, M.D.

The treatment of chronic pain syndromes associated with continuing organic pathology is a most challenging therapeutic situation. Arthritis patients vary in terms of personality types, pain thresholds, different value systems, and good and bad experiences, as do patients in any medical practice. These factors impact on the pain experience and are important in the arthritis patient who has a chronic condition, producing chronic pain.

Patients vary in their response to arthritis. Some patients with minimal rheumatoid arthritis are constantly in pain, and bitter at the total change that has been brought to their lives. At the other extreme are individuals with severe rheumatoid arthritis, severe deformities and serious disability who maintain they never feel pain and that the arthritis is of relatively small consequence. Narcotic tablets are often given to the former while no analgesics are offered or given to the latter.

There are three factors in pain treatment in the patient with arthritis. The first and the one with the highest priority is the treatment of arthritis itself. Acute rheumatoid arthritis is quite painful. One uses rest, heat, braces and non-steroidal anti-inflammatory agents such as aspirin and related compounds. If the inflammation is more chronic and progressive, one uses slower acting drugs such as gold or penicillamine. Selected inflamed joints respond quite well to intra-articular injections of steroids.

The second approach to pain in arthritis patients is the diagnosis and treatment of causes of pain other than arthritis. One must consider extra-articular causes of pain in these patients. For instance an individual with rheumatoid arthritis who complains of "hip pain" frequently may have pain from the trochanteric bursa and not an arthritic hip joint. A simple injection of xylocaine and steroids dramatically relieves the pain.

The third and most difficult is the treatment of the pain itself. While the disease may take some time to clear or in fact may continue unabated, the pain must be relieved. Analgesics may be all that one has to offer and surgery may have to be considered.

A helpful model in viewing the individual accomodating to his arthritis and pain, is that of Kubler-Ross' now well known stages in one's accommodation to the knowledge of impending death: 1) denial 2) anger (why me?) 3) bargaining 4) depression 5) acceptance.[16] These stages apply equally well to the individual who is facing a life of pain from arthritis. Indeed the pain is usually manifested differently in each stage. The first and last of these stages are the easiest to treat with regard to the pain itself. In the early phases, one frequently wishes to support the patient's sense of

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From: Univ. of Arizona Health Sciences Center, Dept. of Psychiatry, Tucson, AZ 85724. Reprint requests to Dr. Shanfield.

denial, since spontaneous recovery in many forms of arthritis is well known. There is frequently little anxiety as the patient waits confidently for the day when the arthritis will lift. Demands for analgesia and the physician's time are usually small. During the anger, the bargaining and the depression stages, management is hardest. A great deal of support is needed along with repeated statements regarding the progress of the disease and its treatment. Persistent depression may respond to antidepressant medication. Analgesia demands may be relatively large, often seeming excessive. Some patients enter the final stage of acceptance themselves and some are facilitated by the physician. Demands on the physician and analgesic medications at this time usually decrease.

There is a major need for the assurance from the physician that he will take care of his patient and see to it that the patient has no unbearable pain. Analgesia will vary from simple tylenol to codeine.

Many patients take 4 to 6 grains of codeine every day without any sign of addiction, function well and feel comfortable when they could not with out this medication. Tolerance in these circumstances is rare. Extenders of the physician are of great value and include psychological counseling, relaxation techniques and arthritis self-help groups.

Pain In Terminal Illness

Stephen S. Cox, M.D.

The term "terminal illness" is usually interpreted to mean cancer. However, many non-neoplastic disease may be as malignant in their clinical course and as painful as cancer and it is reasonable to apply similar methods of pain and symptom control to these diseases. Most of the experience with pain control in terminal disease at Hospice is related to malignancy and the context of these remarks is taken from that area.

In terminal illness one must be aware of psychological issues. One must deal with depression, anxiety, fear, isolation and spiritual needs. A successful approach to these problems areas may obviate the need for potent analgesia. Even the most meticulous attention to a drug program may fail if emotional problems are not fully explored and dealt with. In addition, family needs are extremely important. The patient is often very aware of the family's pain and grieving. This is often reflected in the intensity and frequency of the complaints of the patient's physical symptoms. The environment in which the care of family and patient takes place is also important. A compassionate and caring environment may often diminish the need for potent analgesic medications or tranquilizers. The approach to all of these areas must be individualized using appropriate counseling and pharmaco-therapy as indicated.

The basic control of "permanent" or "intractable" pain which is seen in less than fifty percent of end-stage cancer patients, revolves around the oral morphine suspension. The "Hospice Mix" consists of oral morphine sulfate in a suitable vehicle such as simple syrup or grenadine.[17] Research by Robert Twycross in England has shown that there is no advantage to diamorphine (heroin) over morphine. The cocaine, alcohol and chloroform water that are part of the "Brompton Cocktail" are no longer found to be desirable or useful.[18] The goal of an adequate terminal pain control program is *prevention*. The key elements are to give *enough* medication *often enough*. The pharmacologic action of morphine covers a four hour time period. Thus doses of "Hospice Mix" are given every four hours around the clock indefinitely. The dosage varies and is titrated according to the patient's need. Very often physicians stop short of full control of pain because they are fearful of using adequate dosages because of prior training and prejudice with regard to addiction and tolerance. Doses as low as 2.5mg of Hospice Mix every four hours may be effective. The highest dose that the author is aware of is 400 mg of oral morphine elixir every four hours in a patient in another hospice setting. The concentration of morphine in the elixir can be varied from 1 mg per cc to 10 mg per cc so the volume requirements for the higher doses do not become excessive. One starts the Hospice Mix at a level in proportion to prior medication being used for pain control. If the patient were on parenteral morphine the oral dose is approximately two times as much. An average starting dose, from experience at the hospice, is 10 mg of oral morphine every four hours. Drowziness is common in the first 24 to 48 hours. However, the patient is usually fully functional during this time and the drowziness rapidly recedes after about 72 hours. Tachyphylaxis and tolerance have not be a problem.[19] Doses increase usually because of the increase in pain and not due to decreased efficacy of the medication. It is not uncommon to find that after pain control is achieved and the patient has confidence in the program, doses can be gradually reduced. Some hospice settings routinely include Compazine syrup 2.5mg to 10mg every four hours during the initial use of Hospice Mix therapy to diminish nausea and to alleviate the anxiety that almost inevitably accompanies severe pain.[18] The common use of other psychotropic agents (phenothiazines or antidepressants) depends on the clinical judgement of the managing physician. It is useful to keep in mind that response to pain is complex and includes the actual physical stimulus and the emotional response.

There are a number of adjuvants to Hospice Mix that may help to increase its effectiveness. Simple aspirin seems to have a significant additive effect when given with morphine. Butazolidin or other non-steroidal inflammatory agency may be particularly useful in bone pain. The British use modest doses of steroids (Prednisone 5 mg tid) on a rather routine basis in terminal care because of the sense of well being, increased appetite and generally increased strength that it provides.[19] Anti-tumor therapy may be added when it ill be effective in relieving symptoms without adding the patient's symptom burden. An effective bowel rogram is very crucial with the use of long term high ose narcotics as constipation can be an extremely psetting and painful condition to many patients.

Parenteral therapy may be indicated in certain atients who are simply unable to take oral medication. lorphine sulfate given subcutaneously with a small uage needle is very well tolerated for long periods of me. Using this procedure, problems associated with peated intramuscular injections are not found. In our xperience intravenous medication is never indicated. Ve feel that pain control is important in semi-comatose nd non-verbal patients who have been on significant oses of analgesia prior to becoming obtunded. dditionally we find that the use of atrophine for noisy rminal respirations ('death rattle') is very useful in paring the family discomfort.[20]

There are of course many alternatives to morphine ulfate, including methadone, demerol, dilaudid and lwin. Although we prefer the use of Hospice Mix ecause of the ease of titrating dosage and the oral route, he principle of using "whatever works" should be mployed if for some reason oral or subcutaneous norphine are not considered the drugs of choice.

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Seminars in Endocrinology and Metabolism

Dietary Goals for the United States Do You Agree?

Stephen E. Dippe, M.D.

Editor:
Stephen E. Dippe, M.D.

Many metabolic disorders such as obesity, diabetes mellitus, hyperlipoproteinemia and hypertension can be favorably affected by improved nutrition. However, information on nutrition often does not reach the public or may reach them through unreliable sources. The U.S. Senate has evaluated human nutritional needs and has published dietary goals which could improve the health of the American people.

For about ten years the U.S. Senate Select Committee on Nutrition and Human needs has been determining nutritional needs and priorities for the American people. They originally worked on the prevention of undernutrition and hunger. Over the past few years the

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From: Family Practice Center, Scottsdale Memorial Hospital, 7301 East 4th Street, Scottsdale, AZ 85251

ttention of the committee was directed towards overnutrition and poor nutrition. In February 1977, the U.S. Senate Select Committee on Nutrition and Human Needs published the first edition of the Dietary Goals for the United States. The second edition appeared in December 1977. The goals are:

1. To avoid overweight, consume only as much energy (calories) as is expended: if overweight, decrease energy intake and increase energy expenditure.

Most physicians would find it difficult to fault this goal. How obesity is defined can be debated and even whether obesity alone is a risk factor can be questioned. Not only is obesity associated with elevated triglyceride, glucose, and blood pressure levels but it is also associated with poor self image, lack of physicial exercise, and impaired social goals.

2. Increase the consumption of complex carbohydrates and "naturally occurring" sugars from about 28% of energy intake to about 45% of energy intake.

Over the past 70 years our diet has changed from one that contained more complex carbohydrates to one that contains more refined sugar. The recommendation simply encourages a return to those habits by increasing the consumption of fruits, vegetables, and whole grains. There would have to be changes in meal planning, shopping habits, and restaurant menus. Many foods that contain complex carbohydrates also contain significant quantities of fiber, nutrients, and bulk materials.

3. Reduce the consumption of refined and processed sugars by about 45% to account for about 10% of total energy intake.

The calories from refined or processed sugar are often times termed "empty" calories. More than two-thirds of the total refined sugar is consumed in processed foods and beverages. Therefore, this goal can only be obtained by changing food processing and marketing habits.

4. Reduce overall fat consumption from approximately 40% to about 30% of energy intake.

This goal is primarily related to obesity and cardiovascular disease. The consumption of fat in the American diet has increased approximately 25% since the 1920's. Today fat makes up 40% of our calories. Once again, in food processing fats are added to products. This is especially so in the fast food restaurants and can result in a relatively high consumption of fat (a cheeseburger, french fries, and a milk shake).

5. Reduce saturated fat consumption to account for about 10% of total energy intake; and balance that with polyunsaturated fats, which should account for about 10% of energy intake each.

Encouraging information suggests that the average American diet already contains a lower percentage of saturated fat than 10 or 15 years ago giving this goal a head start. A major stumbling block for the consumer is trying to determine which foods contain saturated fat and which contain unsaturated fat. Even though the fat is vegetable fat it may be hydrogenated or saturated

6. Reduce cholesterol consumption to about 3 mg/day.

This goal is clearly related to vascular disease. The mo concentrated dietary source of cholesterol is found egg yolks, with one egg yolk containing 250mg. Thus, the egg yolks are eliminated or severly restricted in tl diet it may be possible to obtain the stated goal. problem exists with the consumer not knowing tl conentration of cholesterol in various produc especially if egg yolks or animal fat are added.

7. Limit the intake of sodium by reducing the intake salt (sodium choloride) to about 5 g/day.

The controversy concerning sodium intake and its effe on hypertension continues, but is does appear that tl high concentration of salt in the diet may predispo certain individuals to hypertension. Thus, this goal is attempt to reduce the probability of developir hypertension. In order to implement this goal, it wou require placing the concentration of salt on the fo label.

For the average person to meet the goals stated, would have to change his food bought and used follows:

69% more grain products
25% more fruit and vegtables
21% more dried legumes and nuts
10% more milk as skim milk
Replace butter, lard, and saturated vegetable shortening with liquid vegetable oils and soft margarine.
24% fewer eggs
25% less meat, consuming none of the drippings separated fat
59% less visible sugar, syrup, jam, jelly, candy

Certain substances such as fructose and sorbitol a being added to foods or used as sugar substitutes. Th sugars are metabolized to glucose and add calories in th same way as other sugars. There has also been a sligh shift in the sugar available to the American diet fro sucrose to corn syrups.

The following recommendations of the Sele Committee were suggested to help accomplish thes dietary goals.

1. Congress should provide money for a publ education program based on these or similar goals. Th program should reach school children, school foo service workers, recipients of federally funded foo assistance programs, and participants in programs of th Extension Service and the US Department c Agriculture. There should be extensive use of televisio to educate the public.

2. Congress should require food labeling for all food

3. Congress should provide money to the U Department of Agriculture and the Department o Health, Education and Welfare to conduct joint studie and pilot programs in food processing to reduce ris factors in the diet.

4. Congress should increase funding for human utrition in the Department of Agriculture and stablish a committee for coordination of nutrition esearch undertaken by both the Department of griculture and the Department of Health, Education, nd Welfare.

5 These two departments should form a joint ommittee to periodically consider the implications of utritional health concerns on agriculture policy.

These conclusions were reached after considerable estimony by scientists, physicians, consumers, and ven representatives of the food industry and griculture. It basically is a summary of what the itnesses in conjunction with the Select Committee escribed to be essential.

A major obstacle in trying to achieve these goals will e to convince the commercial food industry to undergo ignificant and perhaps drastic changes. Today, certain conomic advantages are gained by using food materials uch as inexpensive sugars and fats. In addition, in order o maintain flavor, color, and texture the food industry as relied on certain basic formulas to maintain onsumer satisfaction.

Estimates for the good budget staying within the ietary goals were almost identical to what is usually pent for food. This data was determined by cutting ack on candy, sweet baked goods, soft drinks, resweetened cereals, and using more chicken, fish, and urkey, legumes and grains.

Questions yet to be addressed are who should or what ody should be responsible for improving the diet of the merican people, or should anybody be responsible, or is he individual himself responsible for his own nutrition? riginally, Americans and other people of the world rew what they ate. However, today many people in the nited States are at the mercy of the food anufacturers. If the manufacturer produces foods that re acceptable for flavor, texture, and color but not cceptable for their nutritional value, should anyone ake responsibility for informing the public.

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Please clip out and sent to:
Stephen E. Dippe, M.D., F.A.C.P.
Family Practice Center
Scottsdale Memorial Hospital
7301 East Fourth Street
Scottsdale, Arizona 85251

Which of the following goals do you agree with?

	Agree	Disagree
1. Avoid Being Overweight	________	________
2. Increase Complex Carbohydrates	________	________
3. Reduce Refined Sugar	________	________
4. Reduce Total Fat	________	________
5. Reduce Saturated Fat	________	________
6. Reduce Cholesterol	________	________
7. Limit Sodium	________	________

Your type of practice

Family Practice or General Practice ________
Internal Medicine ________
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Seminars in Gastroenterology and Liver Disease

What About Nutrition?

Leon A. Rigberg, M.D.

Editors:
Leon Rigberg, M.D.
Stephen Glouberman, M.D.
George Burdick, M.D.

Recent work in the field of hyperalimentation by such physicians as Dudrick, Able, Ansley and Blackburn has shed light on malnutrition in the hospital. In one study 50% of general surgical patients suffered from a moderate to severe form of malnutrition and on the medical wards these figures appeared very similar. Greater assessment of nutritional requirements is possible using a nutritional team composed of a dietician, nurse and physician. Better nutrition improves wound healing and collagen production, increases production of essential proteins, hormones and enzymes and improves the immune system. Catabolic states occur rather quickly in persons with multiple bone traumas, acute infections, surgery and even in normal individuals with "preps" for various x-rays studies and other diagnostic - therapeutic procedures. Reasons for malnutritions are usually inability to eat or low calorie liquids only and inability to replace needed calories intravenously.

The basis for calorie requirements for an adult is 35 to 40 kilocalories per kilogram and for fluid requirements, 35 cc. per kilogram. Protein requirements are about 0.8 grams per kilogram. Lipids are not absolutely required, but linoleic acid represents one to two precent of the total calorie intake. In more understandable terms, this generally means calorie intake between 2700 and 3000 calories in 24 hours. It should always be remembered that the glucose supplies four kilocalories per gram, protein supplies four kilocalories per gram and fat supplies nine kilocalories per gram. Eight of the amino acids are essential, the rest are produced within the body.

Intravenous fluid therapy over the years has primarily been 5% Dextrose and water or variance of 5% Dextrose and Lactated Ringers, Saline and some protein hydrolysates. Presently we talk in terms of "protein sparing therapy," which at this time can be supplied in the form of amino acids with or without Dextrose, with or without intravenous lipids. Amino acids do no stimulate insulin as much as 5% Dextrose solution and therefore allows mobilization of fatty acids for use of calories reducing the amount of nitrogen and protein losses. Amino acids plus 5% Dextrose may come closer to a zero nitrogen balance and with intravenous fat may provide a positive nitrogen balance for a period of five to seven days.

Nutritional requirements are assessed by genera appearance, weight loss, recent medical history and the measurements of skin folds in the mid-triceps region and the circumference of the arm. The male skin fold is 12.5 mm. and the female 16.5 mm., male 25.3 cm. and female 23.2 cm this represents the average circumferencee. Also to be evaluated is the Creatinine height index, which represents muscle metabolism. The measurement of delayed hypersensitivity can be performed as well as the measurement of serum proteins.

Oral alimentation includes chemically defined diets of amino acids and supplements which contain larger peptides and protein hydrolysates, and proteins derived from eggs, milk, meat and soy. Most of these oral diets contain multivitamins, trace minerals, electrolytes, and when mixed properly, contain approximately one calorie per cc. The limiting factor on intake is osmolarity of the oral fluid, which if given rapidly will induce osmotic diarrhea. Some of the oral agents have medium chain triglycerides, some have larger amounts of protein and some must be made with milk, which may produce symptoms of lactase deficiency. With the use of new nasogastric feeding tubes made of silastic and mercury tips (Keofeed), chemically defined diets can be given as a continuous drip for 24 hours and can provide from 2700 to 3000 calories per day if the rate of 125 cc. per hour can be achieved. The volume is slowly increased beginning at 50 cc. per hour, and one third to one half concentration if the small bowel has been resected. Oral route seems to be more beneficial in small bowel adaptation in allowing the small bowel mucosa to undergo hypertrophy more so than through the intravenous route.

Peripheral hyperalimentation utilizes amino acids present varieties which include Feamine II, Travesol and Aminosyn, and provide from 3.5 to 10% protein concentration. The 8.5% amino acid solution provides 12.5 grams of utilizable nitrogen, which represents the equivilent of 78 grams protein per liter. Since this would be given through a peripheral vein, concentrations of glucose higher than 10%, are not utilized because of the sclerosing affect on the veins. Five hundred cc. of 10% glucose can be mixed with any of the three crystalline amino acid solutions, generally a cc. for each cc. Varying

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Precautions: Use with caution in patients with cardiac disease, hepatic or renal impairment. Concurrent administration with certain antibiotics, i.e., clindamycin, erythromycin, troleandomycin, may result in higher serum levels of theophylline. Plasma prothrombin and factor V may increase, but any clinical effect is likely to be small. Metabolites of guaifenesin may contribute to increased urinary 5-hydroxyindoleacetic acid readings, when determined with nitrosonaphthol reagent. Safe use in pregnancy has not been established. Use in case of pregnancy only when clearly needed.

Adverse Reactions: Theophylline may exert some stimulating effect on the central nervous system. Its administration may cause local irritation of the gastric mucosa, with possible gastric discomfort, nausea, and vomiting. The frequency of adverse reactions is related to the serum theophylline level and is not usually a problem at serum theophylline levels below 20 mcg/ml.

How Supplied: Capsules in bottles of 100 and 1000 and unit-dose packs of 100; Liquid in bottles of 1 pint and 1 gallon.

See package insert for complete prescribing information.

Mead Johnson PHARMACEUTICAL DIVISION

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amounts of electrolytes are added and this can be given with an intravenous *lipid* solution through a "Y" type adapter. The lipids should not provide more than 60% of the total calories in 24 hours. Intravenous lipids have been implicated in anemias, hypo and hyper coagulation states and elevated triglyceride levels. At present there are two lipid infusions, one made of soybean oil and the second made of safflower oil. Approximately 2,000 to 2500 calories can be provided through the peripheral vein route and may be sufficient in producing zero nitrogen balance or a positive nitrogen balance.

Indications for TPN (total Peripheral Nutrition) include malabsorption, malnutrition, diarrhea, intestinal obstruction, ulcerative colitis, granulomatous colitis, pancreatitis, anorexia nervosa, intestinal fistulas, renal diseases, some varieties of liver failure, multiple trauma, burns, and as an adjunct in cancer therapy. TPN is utilized when higher concentrations of glucose are required. One can provide sufficient non-protein calories with the utilization of 50 to 70 percent glucose and water without producing sclerosis of the vein. The glucose can be infused at a constant flow rate to avoid fluctuations in blood sugar. With the use of 8.5% crystalline amino acids and equal amounts of 50% Dextrose and water, one can supply 850 calories per liter. The usual amounts of three liters per day supplies a total of approximately 117 grams of protein, 18.75 grams of nitrogen and 2,550 calories. Further calories can be supplied with peripheral intravenous lipids, having one calorie per cc. Minerals, electrolytes, and insulin are added to the amino acid solutions. One must be able to follow blood electrolytes, glucose, calcium, mangnesium, and phosphorus in order to determine the necessary changes. Each hospital should establish flow sheets to be used when the patients are receiving TPN. TPN can treat liver and kidney failure and at present "Freamine E" is the solution available, which contains 13 grams of high value protein made up of essential amino acids. This is the counterpart of the Giordano-Giovionetti diet, which contains 20 grams of protein containing essential amino acids which utilizes urea to recycle and provide proteins.

The preparation of TPN should be in the pharmacy using sterile technique, a laminer flow hood, and then transportation to the needed region in the hospital and stored in refrigeration units. The hypertonic glucose solution is a good medium for growth of bacteria and fungus and the solution should be cultured immediately, if sepsis is suspected. The most common fungus involved is Candida and the most common bacteria are Staph and Strept. Dressing changes should be approximately three times per week. New tubing should be utilized with each liter of hyperalimentation fluid. Catheters, once in place without any complications, can be left in for 30 to 60 days and even longer with the new silastic catheters. Home use of TPN is available for persons with chronic fistula drainage, carcinomas, obstructions, and requires training by the patient, education from the nursing staff, and a portable infusion pump. The patient can be trained to utilize approximately 2,000 calories within a 12 hour period of time, allowing himself freedom in activity during 12 hours of the day. Complications of TPN are basically those of the subclavian catheter. Pneumothorax, hemothorax, puncture of the subclavian artery, thrombosis, emboli and infections are some of the potential complications of subclavian catheterization. The average of infections is reported from 10 to 50 percent, however, of those culture positive, only 7 to 10 percent are found. Possible metabolic complications include hyperosmolar states, hyperglycemia, azotemia, deficiencies of phosphorus, calcium, magnesium, vitamin and fatty acids.

Conclusion

Each physician should be responsible for evaluation of his patients nutritional requirements. Whether or not simple glucose or protein replacement is indicated or more aggressive oral or intravenous alimentation is indicated, the physician in charge should be aware of basic calorie needs of an individual, calorie utilization during periods of stress, and calorie deprivations during common medical diagnostic studies. If a more intense program for evaluation is necessary, it is recommended that nutritional teams be available from the hopsital for consultations to provide a greater nutritional assessment. Generally, the nourishment of the patient is not considered until late in the course of his hospital stay and by then wound healing is impaired and infection and catabolism have already occurred. The hopes of this paper are such that nutrition may well be thought of early in the hospital course and appropriate measures be carried out as soon as possible. Several courses on hyperalimentation are available thoughout the United States and are highly recommended to all physicians for a greater understanding of basic nutrition and modalities of replacement.

Tentorial Meningioma Diagnosed by Coronal Plane Computed Tomography

Enrique L. Labadie, M.D.
Michael S. Smith, M.D.

Editors:
Betty F. Ball, M.D.
Barry Hendin, M.D.
Lawrence Z. Stern, M.D.

Introduction

Tentorial meningiomas comprise about 3-5 percent of all meningiomas (Barrows-Harter 1962) and about 15 percent of all posterior fossa meningiomas (Russell-Bucy 1953). Because of their location, tentorial meningiomas can behave as either supratentorial or infratentorial mass lesions; therefore, precise clinical diagnosis is difficult. Indeed, "tentorial meningiomas leave a lasting impression when discovered at the time of radiography or surgery in a patient suspected of having a totally unrelated condition" (Barrows-Harter 1962). We wish to report a case of tentorial meningioma in whic the preoperative diagnosis was greatly facilitated by us of computed tomography in the coronal plane.

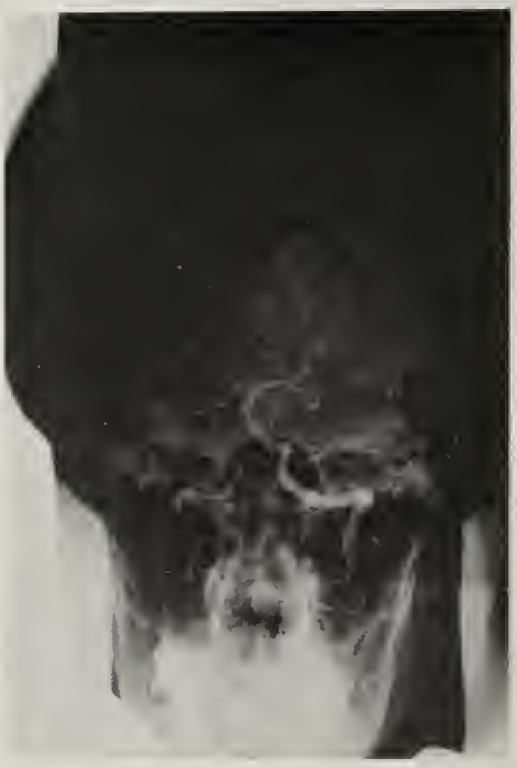

Figure 1

Case Report

A 50-year-old chemist presented with a chie complaint of left temporal headaches of one yea duration which were refractory to analgesics. Si months prior to admission the patient developed readin difficulty, as "the written words made no sense to him. In the two months preceding admission, his wife note progressive deterioration of his memory to the poin that the patient even had difficulty recalling her name

On examination, the vital signs were normal and th general physical examination was unremarkable Neurologically, the patient was fully oriented wit impaired recent memory but intact remote memory. H could perform serial sevens correctly, but he otherwis calculated poorly. In addition, he showed a nomina aphasia with dyslexia, moderate dysgraphia, and a sligh right central facial paresis. The remainder of the moto testing was normal. There was no sensory or cerebella deficits, and the reflexes were symmetrical with the lef plantar reflex being flexor and the right one equivocal

Admission laboratory values were normal, as well a the skull and chest x-ray films. Electroencephalogra revealed leftsided frontotemporal slowing, and a isotope brain scan showed a large, deep, leftside parietal mass. Angiography showed a large, profusel vascularized tumor, supplied from the left diencephali and posterior thalamic circulation (Figure 1).

The presence of identifiable meningohypophysea vessels and diffuse staining led to the tentativ radiographic diagnosis of meningioma. A compute tomography scanning was then performed in both th horizontal and coronal planes with contras

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From: Veterans Administration Hospital, Neurology Dept. and Univ. of Arizona Health Sciences Center, Tucson, AZ 85724. Reprint request to Dr. Labadie, Neuro. Dept., VA Hospital, Tucson, AZ 85723.

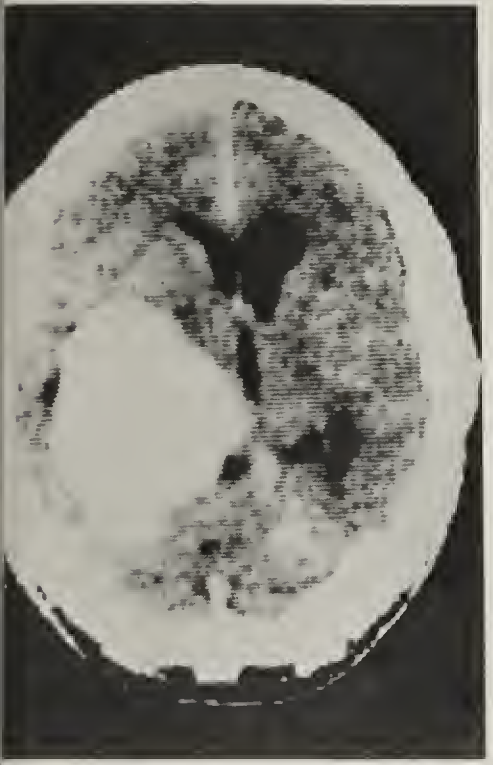

Figure 2

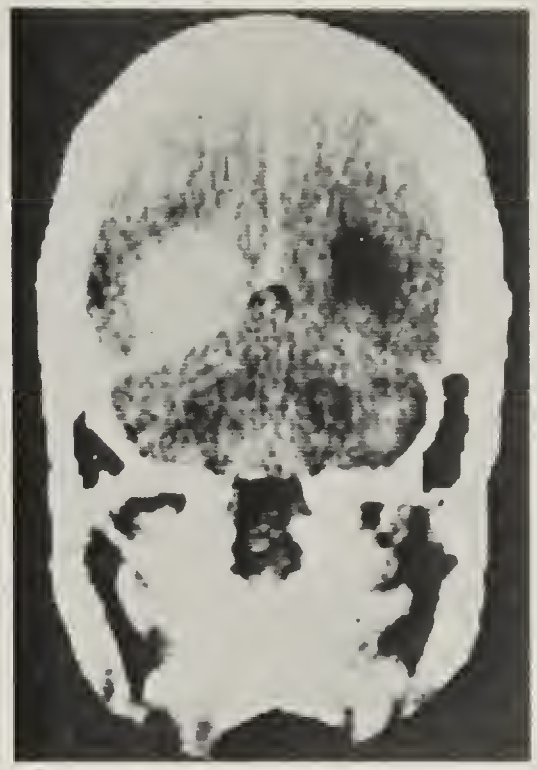

Figure 3a

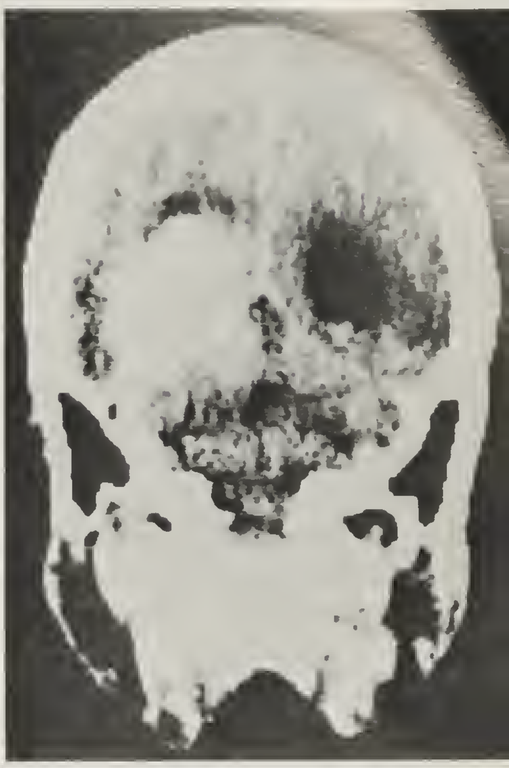

Figure 3b

enhancement. This test clearly revealed a mass localized in the tentorium on the left side and with extension above and below the tentorial surface. Configuration, location and marked contrast enhancement was consistent with the radiographic diagnosis of tentorial meningioma (Figure 2-3).

Ten days after admission, the patient underwent craniotomy with subtotal resection of the tumor. Neuropathological examination verified the tumor to be a meningioma. Postoperatively, the patient's right homonymous hemianopsia cleared and the facial paresis improved. The postoperative computed tomography scan is shown in Fig. 3c.. At discharge, four weeks later, the patient has a mild nominal aphasia with his slight residual deficits pertaining only to his abstract chemistry knowledge. The constructional apraxia had disappeared entirely. As the residual tumor was felt to be surgically inaccessible, the patient underwent radiation therapy to the remaining tumor site.

Discussion:

Barrows[1] divided the presentation of tentorial meningiomas into two types depending upon whether the growth was supratentorial or infratentorial. His supratentorial meningiomas presented with temporal or occipital lobe signs, homonymous hemianopsias, papilledema, hemiparesis, and seizures being common complaints.

Infratentorial meningiomas tended to present in one of three manners: subacute obstructive hydrocephalus with headache, papilledema, and prominent ataxia; chronic indolent mental and gait disturbances; and trigeminal nerve dysfunction causing paresthesias or dysesthesias.

Our patient presented mostly with dysfunctions of the temporo-parieto-occipital areas, due to the large supratentorial mass component. The coronal plane computed tomography scan clearly demonstrated infratentorial tumor mass extension.

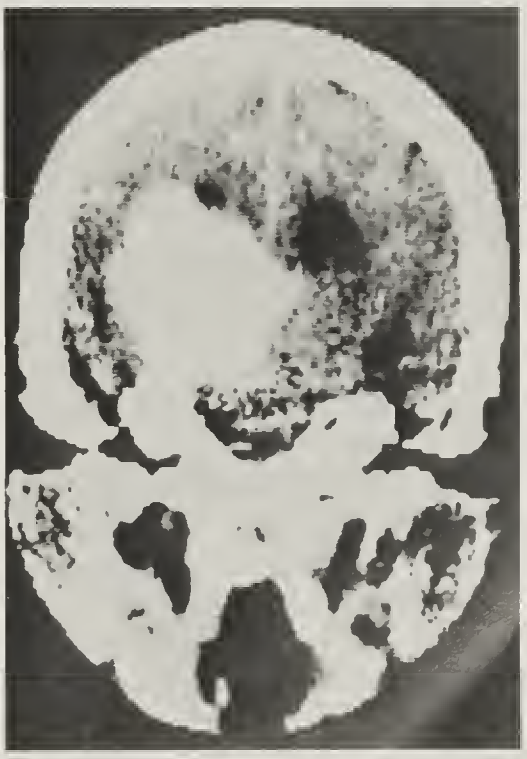

Figure 3c

This case illustrates the diagnostic value provided by multiple plane computed tomography for those mass lesions situated near or along the tentorium cerebelli.

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Obstetrics and Gynecology

Wrongful Life: An Obstetrical Dilemma
Part II

Donald J. Ziehm, M.D.

Donald J. Ziehm, M.D., Editor

Important Developments Cited in Continuing Saga of Birth Defect Lawsuits

In the last issue [Philip Reilly] discussed briefly the history of "wrongful life" litigation, lawsuits brought by parents of infants with birth defects against physicians who had, allegedly, failed to warn the pregnant woman of special risks to her fetus. During the last five years a growing number of courts have permitted lawsuits to be brought by parents to recover damanges for the extraordinary costs of caring for a "defective" child, but the courts are very uncertain about whether to permit parents to recover for the incidental costs of rearing the child or for their own emotional needs.

On December 28, 1978 the New York Court of Appeals, the highest court in that state and the most influential state court in the nation, decided the damage issues in two wrongful life cases, Becker v. Schwartz and Park v. Chessin. in Becker the parents of a child born with Down's Syndrome sued their obstetrician for failure to inform them of the increased risk of bearing such a child to women over thirty-five and for not informing them that amniocentesis can be used to detect the condition in time to get an abortion. In Park the parents of two children born with infantile polycystic kidney disease sued, claiming that after the birth of the first child their doctors had advised them that the condition was not hereditary when in fact it was an autosomal recessive disease. But for the erroneous advice the parents claimed, they would not have risked a second pregnancy.

These new opinions are important for three reasons: 1) the court rejected a cause of action by defective infants of wrongful life, 2) the court refused to permit a cause of action on behalf of either parent for emotional pain and suffering caused by the birth of a defective child, 3) the court upheld a cause of action for the extraordinary medical and institutional costs incurred on behalf of the child.

The court found two flaws in the infant plaintiffs' injury. As the court put it, there is no "fundemental right of a child to be born as a whole fuctional human being." Second the infants' claims confounded the basic aim of tort law - which is to restore an injured party the the "position he would have occupied but for the negligence of the defendant." The court concluded that only the legislature could confer this right to sue upon the infants.

The court's rationale for rejecting a cause of action by the parents for emotional harm is not completely persuasive. Under New York law the courts limit recovery for emotional injury to situations where there has been a breech of a duty flowing from the defendant to the plaintiff, and there are proven harmful emotional consequences flowing from the breach. The court said that "notwithstanding the birth of a child with an abnormality and certainly dependent upon the extent to the affliction, parents may yet experience a love that even an abnormality can not fully dampen." Thus, held the court, "calculation of damages for plaintiffs' emotional injuries remains too speculative to permit recovery notwithstanding the breach of duty." However, when one considers the anguish suffered by parents of a child with a degenerative neurological disorder like Tay-Sachs disease it is difficult to argue that their love for that child counters their suffering.

Finally, concerning the decision to permit the parents to sue for the special medical and institutional costs incurred on behalf of their children, the court found that "but for the defendants' breach of their duty to advise plainiffs, the latter would not have been required to assume these obligations." Clearly, then, it is now the law in New York that an obstetrician must warn a thirty-seven year old pregnant woman of the risks of bearing a child with Down's Syndrome and the avilability of amniocentesis. Further, there is now strong precedent that physicians who incorrectly diagnose the hereditary nature of a congenital defect may be sued by patients who rely on that diagnosis and give birth to a child with a serious defect or disease.

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Oncology

Epidermal Skin Cancer

Robert Allen Schwartz, M.D., M.P.H.

Editor:
David Alberts, M.D.

Summary

Skin cancer is the most common type of cancer. Most types of skin cancer are readily curable early, before cosmetic deformities or actual metastatic disease occur. Basal cell and squamous cell carcinomas are the most common types of skin cancer; melanoma is less common. All three types are epidermal in origin and are linked with ultraviolet light (UVL) — induced carcinogenesis. If physicians and the public were aware of the irreversible cutaneous damage from UVL exposure, ranging from premature aging to precancerous lesions and cutaneous malignancies, a strict regimen of common sense sun protection would be used with enthusiasm. Perhaps people with light complexions should adopt the motto, "natural coloring is beautiful."

It is important for the practitioner to correlate the skin diagnosis both clinically and histologically. When a patient with skin cancer is seen, predisposing carcinogenic factors should be considered. Although usually UVL is the offender, viral and chemical agents as well as genetic and acquired predispositions should be stressed in order to identify high-risk populations. The examination and regular reevaluation of other cutaneous surfaces and, at times, visceral organs when they have been similarly exposed to carcinogens is especially important.

Introduction

Skin cancer probably represents the most common form of cancer in the United States and western Europe.[1,2] Here in Arizona, basal and squamous cell carcinomas are abundant on sun-exposed areas of the body. A third type of cutaneous cancer, which is among the most malignant, is melanoma. This cancer is uncommon, but it also tends to occur on sun-exposed areas of the body and, here in Arizona, as in Queensland, Australia, it is probably much increased in incidence compared with other parts of the world. Queensland, Australia has the highest incidence of melanoma in the world with 31 cases per 100,000 population. However, there is data recently submitted for publication from Dr. Michael M. Schreiber of Tucson that Arizona may, in fact, have an incidence approaching or equal to that of Queensland. Because skin cancer is so common, every physician should be aware of the danger of skin cancer and be able to recognize it. Furthermore, regular skin evaluation in patients exposed to carcinogenic stimuli, including ultraviolet light, represents an important cancer screen and patients should be informed that constant exposure to the sunlight results not only in skin cancer but also premature and unsightly aging of the skin. Our ancestors clearly protected themselves from the sun. Unfortunately, in Arizona as well as in other areas of our country, the modern day trend is to deliberately expose oneself all year round to the sun on the inaccurate premise that a tanned skin is somehow healthier than an untanned skin.

There are some differences in frequency of occurrence of particular types of skin cancers with different races. In Whites, the most common skin cancer is a basal cell epithelioma (BCE). BCE is between two times and ten times more common than the squamous cell carcinoma of the skin. In Blacks, the opposite is true, but the squamous cell carcinomas tend to develop on areas not chronically exposed to ultraviolet light (UVL). Approximately 85% of melanomas in Blacks are seen on the palms and soles. Interestingly, the BCE has the same distribution anatomically in both Whites and Blacks, whereas the squamous cell carcinoma tends to develop in sites of old scars in Blacks. This suggests that squamous cell carcinomas in Blacks may be due to factors other than exposure to UVL. I will discuss other carcinogenic stimuli in the clinical discussion of these particular tumors as well as other clearly non-ultraviolet-induced skin tumors.

In addition to the skin cancers already mentioned, cutaneous sarcomas and lymphomas are occasionally also noted. The best example of the former is Kaposi's sarcoma and of the latter, mycosis fungoides.

In this paper I will not attempt to discuss these tumors, cutaneous markers of internal malignancy, or cutaneous metastatic disease.

Squamous Cell Carcinomas and Precarcinomas

Squamous cell carcinoma of the skin is the second most common skin cancer in Whites and probably the

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From: Univ. of Arizona Health Sciences Center, Sect. of Dermatology, Dept. of Internal Medicine, 1501 N. Campbell Ave., Tucson, AZ 85724.

ɔst common among Blacks and Orientals. It can arise rom precursor lesions or it may occur de novo. The spectrum of dysplasia, in situ carcinoma, and invasive carcinomas when it occurs in the skin is probably analogous to the pattern of changes seen in the uterine cervix. Luckily, most squamous cell neoplasms can be detected early and treated in their premalignant or preinvasive stage.

Actinic Keratoses

The most common premalignant cutaneous lesion in Whites is undoubtedly the actinic keratosis. This is also called solar or senile keratosis. These lesions are really dysplastic areas of skin and are seen in virtually all elderly light-complexioned people as skin-colored to reddish-brown, ill-defined areas on the sun-exposed surface varying in size from pinhead to several centimeters. These lesions are often better palpated as a mild roughening of the skin. It is said that induration, erythema, erosion, or increase in size should alert the clinician to the possibility that these may have undergone a transition to invasive squamous cell carcinoma. Sometimes these lesions have an extremely unusual growth pattern, so as to appear as hornlike projections. Such lesions are called cutaneous horns. A lesion analogous to the cutaneous actinic keratosis appears on the lip as an actinic cheilitis and should be looked for in anyone with actinic damage.

Although squamous cell carcinoma may arise in approximately 10% of untreated actinic keratoses, such lesions almost never metastasize or become locally aggressive. However, on the mucous membrane (such as on the lip) the metastatic potential is significant.

Bowen's Disease

Bowen's disease, or squamous cell carcinoma in situ of the skin, was originally described by Dr. John T. Bowen in 1912 who noted these lesions and their "eminent" malignant potential. Persons with Bowen's disease tend to have single lesions anywhere on the body which present as well-demarcated, scaly, sometimes hyperkeratotic papules, macules, or plaques. On the nail bed, a clue to the diagnosis may be chronic periungual scaling and crusting with nail discoloration. Such lesions, when persistent, should be biopsied. On the mucous membranes, Bowen's disease may appear verrucous or leukoplakic, or as a velvety or reddish plaque. The latter pattern is often called an erythroplasia and will be discussed separately. Bowen's disease on sun-exposed areas may be sun induced; when it occurs on areas not exposed to the sun, the suspicion of arsenic ingestion.and its associated visceral malignancies should be raised. Also worth noting are a number of wart-like variants, some of which can be confused with common virus-induced warts or seborrheic keratoses. In addition, the squamous cell carcinoma may present as dark, slightly velvety plaques in genital areas. In situ lesions may also develop in a number of preexisting skin eruptions, scars, and inflammatory processes. In these, the danger of progression to frank squamous cell carcinoma is more serious.

Although Bowen's disease was once thought to be a marker of internal malignancy, the relationship (other than that already mentioned with arsenic) is currently very much in doubt. Certainly, when squamous cell carcinoma of the skin is found on non-sun-exposed areas, an evaluation for other carcinogenic stimuli would appear in order. If it occurs in the genital area in women of childbearing age, it may be wise to consider possible carcinogenic stimuli which could involve other female genital tract epithelium. There has been a reported increase in genital tract malignancy in these women.

Erythroplasia

Erythroplasia, often associated with the name Queyrat, refers to squamous cell carcinoma in situ or Bowen's disease on the mucous membrane, often on the penis where it may present with a velvety bright-red plaque. Probably more important than the erythroplasia on the penis is that seen in the oral cavity. Such lesions may progress into squamous cell carcinoma of the oral cavity, which as an ominous prognosis if not detected early.

Oral squamous carcinoma correlates strongly with tobacco smoking and chewing as well as alcohol intake. It tends to be seen in persons in their 60's and 70's. In the alcoholic, lack of vitamins may induce atrophy as a possible carcinogenic stimulus, although this is not totally clear. However, it is clear that oral squamous cell carcinoma is a significant killer, accounting for about 5% of neoplasms in men and 2% in women. It is curable if detected early in its in situ, erythroplastic form. Unfortunately, this is not usually the case and the five-year survival averages about 30% in such patients.

Although traditionally one emphasizes the premalignant potential of leukoplakia in the oral cavity, erythroplasia (that is red instead of white plaques) is probably much more important. It has been suggested that most leukoplakic areas are benign and, in fact, only 2% of oral cancer is primarily leukoplakic. Any erythroplasia, especially if on the oral cavity, should be biopsied if the etiology is not obvious or if it doesn't resolve within two weeks. In addition, since people most at risk are those with society's worst drug-abuse habits, alcohol and tobacco use, these persons deserve regular oral cavity evaluation for this all too common killer.

Arsenical Keratoses

Exposure to arsenic provides a good model for the study of chemical carcinogenesis.[3] Arsenic ingestion appears not uncommonly due to medicinal, environmental, and occupational exposure. Skin lesions often develop at the sites of trauma and friction, especially on the palms and soles, as discrete, often symmetrical, tiny papules, which may group together as verrucoid plaques. In addition, there may be areas of hyperpigmentation, often around the axilla, with islands of normal or decreased pigmentation creating a rain-drop-like appearance. This hyperpigmentation, which nay be marked, sometimes resembles that of Addison's lisease. There may be also a wide diagonal band of oigment or white strips across all or some of the nails. These white lines are called Mees' lines. In addition, the nultiple lesions of Bowen's disease on the trunk and imbs may be seen and there may also be a diffuse lopecia and peripheral circulatory disorders. Such stigmata should certainly alert the clinician to the ossibility of arsenic exposure.

Such exposure can be cryptic and one must consider a wide range of possible environmental sources such as water supplies, certain medicines such as Fowler's solution which was used for many years for a number of utaneous diseases, and old medicinal "tonics." Occupationally, arsenic is still a problem in some smelting, metallurgy, and agricultural areas. Interestingly, a recent source of dietary arsenic was identified in two patients with arsenical keratoses who had eaten a lot of kelp as a health food supplement. Certainly if arsenic exposure can be documented, one must be concerned about the possibility of the development of a visceral malignancy. Lesions parallel to those seen on the skin may develop in the respiratory, gastrointestinal, or genitourinary tract, and thus, arsenical keratoses should be viewed as a cutaneous marker of possible internal malignancy.

Squamous Cell Carcinoma

As mentioned previously, squamous cell carcinoma (SCC) represents the most common type of skin cancer in many parts of the world. Although it can be seen anywhere in the skin or the mucous membrane, it tends to rise from premalignant or in situ squamous cell carcinomas such as actinic keratoses, arsenical keratoses, Bowen's disease, and chronic cutaneous radiation damage. Occasionally it will arise de novo. In dermatologic practice, most of these lesions do result from actinic damage. However, patients with the chronic osteomyelitis or chronic scarring from whatever cause (including chronic cutaneous lupus erythematosus and the deep fungal infections) should be watched very carefully. The reason is that patients whose disease course is complicated by squamous cell carcinoma tend to have a very high incidence of metastasis. This important association is also seen in a number of genetically determined skin eruptions including epidermolysis bullosa.

The de novo squamous cell carcinoma represents at times a difficult diagnostic challenge. It may be confused with a keratoacanthoma, which is a benign, dome-shaped tumor which develops a central keratotic plug and which also tends to arise rather abruptly on the sun-exposed cutaneous surface. Keratoacanthomas may represent malignancies which have failed but, this is not always the case, since they sometimes progress to actual invasive squamous cell carcinomas.[4, 5] It is important that these lesions be biopsied by a physician familiar with the keratoacanthoma and evaluated by a pathologist who is experienced with the keratoacanthoma. This tumor histologically is indistinguishable from a squamous cell carcinoma.

Another very interesting variant which is being described more and more frequently and which the clinician should be aware of is the verrucous carcinoma. This is a type of squamous cell carcinoma of the skin which may be seen as an elevated warty growth which is locally invasive and rarely metastasizes. Over the past years, these lesions have been reported in the oral cavity, larynx, the penis, the vulva, the vagina, and on the scrotum. They are also seen on the bottom of the foot and may represent the malignant transformation of preexisting viral lesions. Certainly the clinician should be alert to this entity and persistent warty growths should be carefully evaluated. It is important to note that the usual pattern of verrucous carcinoma is microscopically well-differentiated; this is clearly distinctive from the more anaplastic verrucous variant of aggressive SCC. In addition, one whould be aware that podophyllin, a commonly used topical therapy for viral warts, alters the histological pattern so that the lesions may apppear as anaplastic SCC.

In passing, I should also mention an entity called epidermodysplasia verruciformis. This autosomal recessive disease usually begins in infancy or childhood. Multiple flat warts are the most common manifestation. These tend to be chronic and widespread. Apparently, some of these warts evolve into premalignant lesions and skin cancers in the teens or later. It is said that about one third of these patients will develop actinic keratoses, Bowen's disease, or squamous cell carcinomas on sun-exposed areas. A delayed hypersensitivity defect in these patients has been suggested by recent reports.

Basal Cell Epithelioma

The basal cell epithelioma (BCE) or basal cell carcinoma is an extremely common skin cancer which usually presents as a dome-shaped nodule on sun-exposed areas, especially the face. Unlike the cutaneous squamous cell carcinoma, the BCE almost never metastasizes, although if left alone it can be not only locally destructive but in addition actually kill by invading through the orbit of the eye into the brain.

The major environmental factor responsible for the development of the BCE is UVL radiation from the sun. Less commonly, x-ray radiation has been implicated. The BCE tends to appear in people with a fair complexion. When it appears after x-ray exposure, it usually appears on hairbearing areas.

There are four common types of basal cell epitheliomas. The most typical is the *nodular* type, which may have a central ulceration. This tends to begin as a smooth, surface nodule, often with a pearly quality to its border and some dilated blood vessels (telangiectasia). Then it tends to grow larger, with central depression and, ultimately, ulceration. Sometimes the borders also have a rolled quality. The pearly traslucency of its borders are due to the uniform tumor cell masses which are well appreciated histologically. This type of BCE may sometimes become pigmented, in which case it may be referred to as a *pigmented* basal cell epithelioma. Pigmented basal cell epithelioma. Pigmented basal cell epitheliomas may at first glance resemble melanomas. Another type of BCE is the *superficial* BCE, which tends to appear as an area of eczema or dermatitis, sometimes with a fine, slender, somewhat pearly border nodulation. At times, the surface may appear superficially ulcerated. Superficial basal cell epitheliomas may occur as multiple lesions in association with the basal cell nevus syndrome. The fourth type of basal cell epithelioma is the so-called *sclerosing* basal cell epithelioma, which can easily be mistaken for a scar with indistinct borders.[6] Recognition of this lesion is particularly important because it tends to appear in a younger age group than the other basal cell epitheliomas. A sclerosing component may appear in nodular BCE and may have a tremendous destructive potential. A lesion on the nose, for example, may erode deeply, resulting in disfigurement upon surgical removal. It needs to be stressed that sclerosing components of nodular lesions and sclerosing BCEs themselves must be viewed with concern and evaluated by a clinician experienced with skin cancer. This is especially true since a newly described tumor, the desmoplastic trichoepithelioma, has recently been separated clinically and histologically from the BCE.

Unusual locations for the basal cell epithelioma include the vulva, the scrotum, and the nipples. At such sites, these lesions may be overlooked. In addition when one sees these lesions at these sites, one might consider x-ray exposure or other carcinogenic influences, since ultraviolet light exposure is infrequent there. A history of x-ray exposure without adequate shielding in areas such as adjacent to the thyroid or breast should prompt an examination for possible thyroid or breast cancer.[7]

The basal cell epithelioma may also be seen in the syndrome called the basal cell nevus syndrome (BCEN). This is an autosomal dominant highly penetrant disorder which may appear at birth or shortly thereafter; however, it can appear at any age. This syndrome was so named because of the original impression that these mole-like facial nodules were melanocytic nevi or moles. They are, in fact, basal cell epitheliomas from the beginning. In addition, one may see superficial basal cell epitheliomas on the back, which should alert one to the possibility of this syndrome. Approximately one half of these patients have distinctive pits on their hands and feet appearing as irregularly shaped depressions approximately 1—3 mm in depth. Such lesions may actually evolve into basal cell epitheliomas. These pits are considered to be pathognomonic of the syndrome. Other findings of the syndrome include jaw cysts, which may become infected, resulting in presentation to a physician. A full description of the syndrome and its complications appears elsewhere;[2, 8, 9] however, it is important for a physician to be aware of its existence.

Actually, multiple basal cell epitheliomas on the sun-exposed areas are certainly not surprising. However such multiple lesions or in fact any basal cell epitheliomas in a young patient should at least raise the clinician's suspicion with regard to the BCEN syndrome Basex syndrome, xeroderma pigmentosum, and the Torre syndrome. The latter, originally described by a prominent New York dermatologist, associates the BCE with histologic differentiation toward sebaceous glands and sometimes keratoacanthomas with low-grade visceral adenocarcinomas. Other unusual presentations of the BCE include the fibroepithelioma, the cystic BCE the linear basal cell nevus, Basex syndrome of multiple "ice-pick" marks on the elbows and dorsal hands with basal cell epitheliomas, and basal cell epithelioma occurring in the nevus sebaceous. These unusual conditions do not merit discussion here, although with regard to the cystic BCE, it should be stressed that unusual lesions, whether cystic or otherwise, merit biopsy after evaluation by a physician experienced in cutaneous oncology. In addition, the acquired predisposition to SCC and BCE (especially the former) in immunosuppressed renal transplant patients on skin actinically damaged, has been recently and convincingly documented.

Melanoma

Melanoma is the third type of skin cancer to be discussed. Melanoma or malignant melanoma is the most dangerous type of skin cancer, although when detected early it may be completely curable. There are four types of cutaneous melanoma which merit discussion here.

The first is the *lentigo malignant melanoma.* This type of melanoma which may appear as a premelanoma (lentigo maligna or Hutchinson's freckle) characteristically begins in its premalignant or preinvasive form as a small brownish macule which resembles an ordinary senile lentigo. It gradually increases in size with the border becoming markedly irregular and the color displaying some variation from tan to brown to black to red. There may be areas of cutaneous regression. Sometimes there is a stippled hyperpigmentation noted in this lesion. When the lentigo maligna stops growing horizontally and grows vertically through the epidermis into the underlying dermis, it is no longer designated a precancerous lentigo maligna, but instead, a lentigo malignant melanoma. Fortunately, lentigo malignant melanomas still are associated with a good prognosis. These melanomas tend to appear in elderly people and are probably more common in women than in men, with often a long history at presentation of up to 40 years. They tend to be most associated with sun-induced damage and commonly appear on the face.

The second type of melanoma is the *superficial spreading melanoma*. It probably represents the most common type of melanoma and is most common on the upper back. These tumors are somewhat irregular in outline although less so than lentigo malignant melanomas;

both may have notched borders. Nodulation may develop if the tumor cells grow vertically rather than horizontally, an omnious clincial sign.

The third type, the *nodular melanoma*, appears without an antecedent macule or plaque. Although it sometimes is skin colored and hence called amelanotic, more often it is dark brown or black. The grayish or blue hue sometimes seen reflects the melanoma cells occurring deep in the dermis. Nodular melanomas are often confused with many other lesions, including melanocytic nevi, stuck-on appearing warty nodules called seborrheic keratoses, and dome-shaped smooth nodules called dermatofibromas.

The fourth type of cutaneous melanoma is the *acral-lentiginous melanoma*, which tends to appear on the palms, soles, or fingers, especially in dark-skinned people. It is very important to be aware of this because biopsies often show a simple "melanocytic hyperplasia without evidence of melanoma." Rarely, melanoma may be polypoid.

There is a group of patients predisposed to the development of melanomas. These include up to one half of the patients with xeroderma pigmentosum, a number of patients with so-called bathing trunk nevi, and the recently described syndrome of multiple acquired familial melanomas in association with prominent melanocytic nevi which are variable in size, outline, and in color combination. This familial tendency to malignant melanoma has been named by Dr. Wallace Clark, Jr., and his associates as the "B-K mole syndrome" in honor of two young patients with this syndrome who together had seven primary melanomas between them.[10] Xeroderma pigmentosum is a rare hereditary disorder characterized by a freckling with actinic keratoses, keratoacanthomas, basal cell epitheliomas, squamous cell carcinomas, and melanomas developing as early as childhood. It is due to enzyme defects in DNA repair during DNA replication or post-replication. In addition, redheaded frecklers and possibly carriers of this genetic trait may constitute a group more prone to DNA repair deficits than others (without xeroderma pigmentosum) and, therefore, are more likely to develop skin cancers including melanomas.

Management Considerations

The management of skin cancer is beyond the scope of this presentation. Recent advances in the chemotherapeutic and immunotherapeutic approaches which were pioneered by Dr. E. Klein and associates at Roswell Park Memorial Institute are summarized elsewhere.[11] [12] The familiarity of the physician with the variety of techniques which may be used may be more important in some cases than the actual selection of the therapeutic modality. Rather than stressing therapy, it is more appropriate in this context to stress prevention, since, for the most part, the squamous cell carcinomas and basal cell epitheliomas and a good number of malignant melanomas are ultraviolet light induced and, thus, preventible. It requires the common sense measures used by our ancestors to protect against the premature aging and carcinogenic influence of ultraviolet light exposure. Such common sense ideas include deliberate avoidance of sun exposure rather than deliberate exposure. People should appreciate the fact that the skin changes and damage resulting from sun exposure are not desirable and that natural skin color is probably the most beautiful.

An intelligent sun protection program involves literal avoidance of sun exposure whenever possible combined with protective clothing and sun screen use when exposure may be necessary as in an occupational situation or when enjoying activities which occur under direct sun light. There are multiple sun preparations available to protect against what are felt to be the most likely carcinogenic wavelengths from ultraviolet light exposure. It is very important to emphasize that those most at risk of developing skin cancer are those with predisposing rare diseses already mentioned, renal transplant patients, those that are light complexioned, and those with previous history of skin cancer. Although these people obviously require special surveillance, it is equally important that the general public be aware of the premature aging and carcinogenic influence of the sun's rays.

It should be remembered that the diagnosis of skin cancer requires not only a gross but also a microscopic pathologic view. It is essential that an experienced skin pathologist evaluate the specimens whether he is a dermatologist or not, since there are a number of benign conditions which may simulate skin cancers both clinically and histologically. Granulomatous diseases including tuberculosis, syphilis, and North American blastomycosis may closely resemble either a squamous cell carcinoma or a basal cell epithelioma either clinically or histologically. Genital warts treated with podophyllin may be histologically indistinguishable from SCC. In fact, so called juvenile melanomas, which are benign, compound melanocytic nevi, may be confused with melanomas. Thus, it is important for a physician to be well trained if he is treating skin cancers, just as it is important for the pathologist who reads the microscopic sections to be conversant with many of the unusual cutaneous findings which occur. Worth emphasizing is the fact that the gross pathology (that is, the patient's lesions) need to be considered together with the microscopic findings because skin cancer and its proper management require such correlation.

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Psychiatric Disorders

Social and Emotional Determinants of the Death Process

Stephen B. Shanfield, M.D.

The concepts felt to be most useful in the understanding of the dying and their families will be discussed. These ideas have arisen in the context of two years of experience as a psychiatric consultant to a hospice - a facility for the humane care of the terminally ill, and as a psychiatric consultant to a cancer service.[1]

Current Interest In Death

The current popularity of death as a clinical phenomenom is a post World II occurrence and can be accounted for in part by the collective shared experience of massive and absurd death, as in the Holocaust, in Russia in the Gulag Archipelago, in Hiroshima and in Nagasaki. The sharing of these events is a product of a post-industrial nation; radio, television - instant communication. The most recent event in relation to death was the difficult American experience in the Vietnam War, which heralded the mass interest in death and dying.[2]

Death Profession

Physicians are members of one of the death professions.[3] They become professionalized to issues concerning illness, death and loss, which remain at the forefront of their work throughout their careers. Indeed a primary task is to deal with the painful effects of grieving which accompany any loss, so that they can be of help to their patients, and sustain their work in a relatively comfortable state.

Attitudes Towards Death

Thoughts of death, extinction, termination or mortality are very common, particularly during illness and in the survival of the loss of a loved one. Death is also implied in any discontinuity or transition of life, in which one experiences the death of an old part of life and the rebirth of a new aspect of life. In this sense we experience many deaths in our lifetime - the break-up of love affairs, divorce, illness, passage from medical school to residency, from residency to private practice and many more. Change is accompanied by a mourning process, the outcome of which can be creative.[4] It is from these experiences that we mature. We can know of death through experience with transitions and other losses The knowledge of personal death has been termed middle knowledge of death since one can never experience the final moment of death before hand.

The middle grouping of years in adult development, roughly convering 35 to 50 years of age, has been felt to be a time of sense of heightened awareness of decline, a time of vulnerability and a sense of one's mortality.[5] This stage of adult development is important in coming to terms with one's death.

Our attitudes toward death are in part shaped by our experiences with transitions. These attitudes include fear, reluctant acceptance, wish for postponement, denial and welcoming of death.[6] Fear and denial of death as prominent feelings are not universal as has been felt by some in the past. For most, attitudes about death are a matrix with more than one feeling being present. Some of these attitudes may be more prominent depending on age, experience with death and illness, as well as present circumstances. Thus one might be able to accept the possibility of one's death yet not want it to happen at a partiuclar time in one's life. This is not uncommon and is seen in many people who have experienced serious illness.[7] For instance, a thirty-five year old divorced mother of two with breast cancer, although able to

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From: Univ. of Arizona Health Sciences Center, Dept. of Psychiatry, Tucson, AZ 85724 ccept her death, wanted to live so that she could take are of her young children until they were old enough to o so on thier own. When one is depressed and feeling epleted the wish for or welcoming of death may be rominent. Many elderly individuals welcome the ossibility of their own death. They feel they have little o look forward to or to live for.

Stage Theory

The well known theory of Kubler-Ross of stages of cceptance of death - denial, anger, bargaining, epression and acceptance has been important in helping o focus and clarify the difficulties of the terminal rocess.[8] However, as with any stage theory, articularly in adulthood, it suffers from the problems hat not all have these stages, nor if present, are the tages necessarily in any particular order. The theory is helpful in providing a sense of the range of issues that rise when facing one's death. Parenthetically, the eelings suggested by Kubler-Ross can accompany any oss or separation.

Grief

The person who is dying grieves or mourns the nticipated loss of family, the loss of bodyly function and he loss of place in the world. Grief is a normal process in which the individual responds to loss with symptoms not unlike depression.[9,10] Anything which interfere with the state of consciousness, such as medication, can nterfere with the grieving process. Although an upsetting time for the patients, clinical experience reveals that most are able to accept their own deaths.

Families also grieve in an anticipatory fashion. Widows and widowers are at high risk for increased morbidity and mortality in the bereavement period. There appears to be increased risk in the bereavement period when the illness of the spouse is longer than six months. In addition, young widows with children seem to be at high risk for increased morbidity in the bereavement period.[9] Clinical experience at the hospice ndicates that high risk survivors seem to have a marital relationship characterized by constriction of affect, skewed decision-making, an unequal distribution of power, low autonomous functioning of one of the pair and a poor capacity for solving problems as a couple. The physician should identify high risk individuals prior to the death of the loved one in an effort to prevent difficulties in the bereavement period. Often, psychological and social supports as well as an increased altertness for medical problems are needed in the period after the death of a loved one. Also, self-help programs such as "Widow to Widow" can be quite helpful for the bereaved.

Terminality

The point of knowledge of terminality is a psychological crisis for the patient the family. From this moment flows an important series of events. The patient and the family start their active grieving and begin to settle their affairs.[8] The physician provides diagnostic and treatment services designed to maximize the highest quality of life for the patient and family.

In the telling the patient of his or her terminal illness there are three variables; the patient, the family and the doctor.[1] The patient most often has thought of the range of possibilities of his disease and in time will know about his terminal state if not told, from the behavior of those around him.[9] The family may place a great deal of pressure on the physician not to inform the patient, as a way of avoiding the pain of loss and the open acknowledgement of grief. The result is often withdrawal which can cause the patient increased suffering, depression and pain. As a matter of policy many physicians simply tell their patients about their disease and its ramifications. They then ask the patient if, how and who in the family is to be told. This fits in with a recent Gallup poll which reveals that 90% of a random population wish to be told of a fatal illness.[10] Such a discussion preserves the integrity of the relationship. The physician can then provide a setting for the expression of the painful feelings associated with grief for both patient and family, and indeed facilitate this process. We need to know more about grieving styles, which will be of help to the physician in his work with the patient and family.

The assignment of terminality is more common in cancer patients in spite of the fact that cancer accounts for only a quarter of the total deaths. Indeed, cancer has been a cultural metaphor for death. There are a number of diseases which are as fatal as cancer, where there is uncertainty as to when one will die. It is more difficult to assign a death trajectory, which is a probability statement about the course of the disease, to many diseases other than cancer, because death is often from some other treatable cause. Indeed, many of the studies of dying patients have been of dying cancer patients who have a relatively predictable trajectory and usually a chronic course. This allows for an exploration and dealing with death in manner different from other diseases. More information about the dying process in diseases other than cancer would be valuable and helpful to the practicing physician in his dealing with the dying patient and the family.

Denial

Often at the hospice the author is asked to see patients who are "denying" their illness. It is helpful to view denial as a psychological attempt to maintain the world as if an event or series of events did not occur.[14] For the dying patient who is often aware of his illness and its consequences, the denial is rarely total and often it is selective. The patient will avoid discussing death with the family or the doctor in order to avoid a confrontation with his and his family's active grieving. On the other hand the patient may talk openly with the a nursing aide who lends a supportive, non-intrusive ear to the patient's difficulties.

Hospice

Hospice care of the terminally ill and their families has four coordinated components; inpatient care, home care, partial hospitalization and bereavement services.[15] The inpatient unit is a special cancer hospital where all the patients are terminal; and death, dying and grief are openly discussed. Care is for the family as well as the patient. This level of care is coordinated with other components so that the patient may be cared for at home and periodically be admitted briefly to the inpatient unit. The bereaved are followed throughout the illness and high risk survivors are identified.[1] Many physicians are delivering the hospice concept of humane care without actual utilization of the hospice organization. However, the hospice team is expert in dealing with the psychological and social elements in the care of the dying patients and their families, as well as in the relief of symptoms such as pain.

Pain for the dying patient is often more difficult to deal with than death. Oral morphine or "hospice mix" on a fixed dosage schedule is the mainstay of treatment of cancer pain.[16] The relationship between patient and staff as well as the treatment of anxiety and depression are also important in the management of pain. This treatment may include the use of anti-anxiety agents and anitdepressants.[17]

The hospice team can provide expert consultation around the treatment of other difficult symptoms and end stage cancer. Participation in hospice care will allow for greater and more accurate observations of the dying patient and the family.

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Radiology

Case of the Month No. 40

John Siler, M.D.
Glenn C. Cook, M.D.
John Bjelland, M.D.

This female infant was born at thirty-four (34) weeks gestation following premature rupture of membranes She did well until the forth post partem day when, shortly after feeding, she developed irritability, tachypnea, and abdominal distension. Due to rapid clinical deterioration of the patient, a plain film of the abdomen was obtained. What is the diagnosis?

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From: Univ. of Arizona Health Sciences Center, Dept. of Radiology, Tucson, AZ 85724

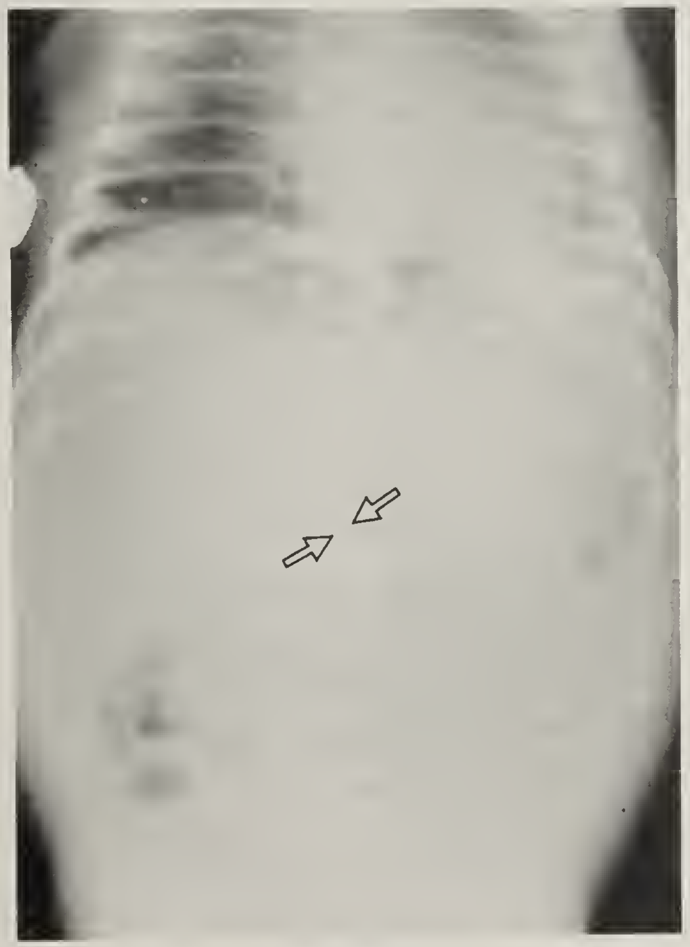

Figure A
AP supine radiograph of the abdomen.

Motorcycle Safety: An Epidemiologic View

Dennis V. Cookro, M.D., MPH

Summary

In 1975, 47 states had helmet-use laws for motorcyclists. As of 1978, 26 states had repealed those laws. Epidemiologic studies done in several of those states indicate that helmet-use is associated with a significant decrease in risk. Arizona Department of Transportation figures show that motorcycle fatalities have increased substantially since repeal of Arizona's helmet-use law. Factors other than helment use that may decrease risks include, rider safety courses, making the motorcycle and rider more conspicuous, and avoiding alcoholic beverages prior to riding.

Historical Background

In 1966, there were only three states with motocycle helmet-use laws. In July 1967, the U.S. Department of Transporation established a number of safety standards, one of which called for the use of helmets and eye protection in the operation of motorcycles. A year later, the Arizona State Legislature passed a helmet-use law which was to become effective in 1969. By 1975, 47 states had passed helmet-use laws.

The Department of Transportation then threatened to decrease highway funds available to states not in compliance with the helmet-use standard. This ultimately resulted in an amendment of the Highway Safety Act in 1976 prohibiting the Secretary of Transportation from requiring that each state enact laws or regulations providing for the use of a helmet by anyone 18 years of age or older. By September 1978, 26 states had repealed their helmet-use requirement. The Arizona Legislature changed its helmet-use law in the same year in which the Congress had removed the possibility of financial penalties. The Arizona helmet-use law now applies only to those motorcycle operators under 18 years of age.

Dr. Cookro is a Preventive Medicine resident at the Univ. of Arizona. He has been assigned to the Arizona Department of Health Services for the past year.

Table 1
Motorcycle-Associated Fatalities in Relation to Motorcycle Registrations, by year, Arizona, 1966-1977

Year	Motorcycle Registrations*	Fatalities	Rate per 1000 Registered Motorcycles
1966	17,349	15	0.86
1967	20,464	34	1.66
1968	25,460	30	1.18
1969	27,699 Helmet Law	15	0.54
1970	34,335	25	0.72
1971	42,884	25	0.58
1972	49.175	41	0.83
1973	56,700	53	0.93
1974	65.018	64	0.98
1975	67,897	63	0.93
1976	60,276 Repeal Late 1976	49	0.81
1977	65,141	77	1.18
1978	68,359	88	1.29

*does not include motorscooters or mopeds
Data from Arizona Department of Transportation Traffic Accident Summaries

Following the repeal of helmet laws in the United States, the rate of fatal motorcycle accidents showed a startling increase.[1] In Arizona, the average fatality rate per 1,000 registered motorcycles during the years 1965-68, prior to the passage of the helmet law was 1.16. The same rate for the years 1969-75, when the helmet law was in force, was 0.82. In fact, during these years, the rate was well below 1.00 for each individual year (see Table 1). The rate for 1977, which was the first full year after repeal of the helmet law, climbed to 1.18 deaths and the rate for 1978 was even higher at 1.19 deaths per 1,000 registered motorcycles.[2]

North Dakota Study

In North Dakota, one of the states which amended its state statute requiring helmets, a two-year study of the impact of the amendment has been conducted by a coalition of several state agencies.[3] Results of that study include the following:

1. Nearly 75% of injuries occurring to the head, face or neck occurred to individuals who were not wearing a helmet at the time the accident took place.
2. There was a highly significant statistcal difference between the injury rate for helmeted and unhelmeted motorcyclists when assessing head, face or neck injury.
3. In approximately 80% of motorcycle fatalities, head or neck injuries were mentioned as the cause of death.
4. Although unhelmeted motorcyclists accounted for about 53% of all the minor or serious only accidents, they accounted for almost 80% of all the critical or fatal accidents. (This led the study group to conclude that the use of a protective helmet is a most important factor in reducing the severity of motorcycle accident injuries, as well as reducing the mortality.
5. Unlicensed operators accounted for slightly over 50% of all motorcycle accidents and fatalities.
6. Eighty percent of all individuals invovled in motorcycle accidents were age 25 or younger, and 75% had five years or less of experience in the operation of a motorcycle.

North Dakota's helmet statute still requires all operators under the age of 18 to wear helmets, as does Arizona's law. Unfortunately, the North Dakota study found that in almost 40% of the accidents occurring to those under the age of 18, no helmet was worn. This demonstrates rather vividly the enforcement problem of a statute requiring mandatory helmet use for those under age 18 only.

One of the most interesting areas the North Dakota study attempted to explore was that of the economic impact of motorcycle injuries and fatalities. Although work in this area is not complete, the study did take a close look at a selected number of motorcycle accidents. Physician and hosptial estimates of the cost of hospitalization or other care were collected for 121 accident cases, and costs for these 121 cases ranged far in excess of $110,000. In Arizona in 1977, there were 3,347 injuries as a reuslt of motorcycle accidents. If we assume the 121 cases in North Dakota were representative of the true picture, then by simple extrpolation, an estimate of the economic impact of hosptial and other care for 3,347 injuries could be well over $3,000,000.

Obviously, motorcycle injuries do have a significant economic effect. If the motorcycle rider dies or becomes permanently disabled, his family must bear tremendous psychological and social, as well as economic, costs.

Other Studies

After repeal of its helmet law, the State of Minnesota experienced and all-time record of 94 motorcycle fatalities.[4] Over 30% of the drivers involved were operating without a valid motorcycle license or their licenses were under suspension or revocation. Over 40% had been drinking. The fatality rate for 1977 (after repeal) was 6.21 deaths per 10,000 registered motorcycles, compared with only 3.98 deaths per 10,000 registered motorcycles in 1976 (helmet law in effect).

The State of Colorado saw a 260% increase in the proportion of motorcycle accidents *with severe head injuries* and an increase in rider fatalities from 14 per 1,000 to 22 per 1,000 riders with repeal of its helmet law.[1]

In Southern California, a study of over 900 motorcycle accidents has shown that riding without a helmet increases the risk of head injury by five times.[1] [5] [6] Th
same study has found *not* one singl
accident in which the wearing of a helme
caused an accident by impairing hearin
or vision, nor did helmets cause any typ
of injury other than minor skin burn
from the chin strap in severe impact
This study contradicts the contention c
opponents of helmet laws that helme
cause more accidents because c
decreased hearing or visibility, an
refutes, as well, the
contention that there are more nec
injuries when helmets are worn.

The "Safe" Rider

There is no doubt that the "safe ride
can decrease his risk of having a
accident. Rider education courses an
safety training in schools (starting at a
early an age as practicable) have th
potential for saving life and limb. Bu
unfortunately, for those who are "saf
riders," a large proporation of fat
accidents may be unavoidable. In Arizon
in 1977, accident reports show that i
almost one-fourth of all motorcycl
accidents, there was no improper drivin
on the part of the motorcycle operator.
the motorcycle operators who had bee
drinking and were involved in fata
accidents are disregarded, then about *ha*
the fatal motorcycle accidents involved n
improper driving. Safe riding, ther
demands that a helmet be worn for thos
"unavoidable" accidents. Additiona
measures that may help to reduce th
"unavoidable" accidents are mor
conspicuous clothing for the rider and us
of lighting on the motorcycle even durin
the daytime.

Arizona Motorcycle Facts

In Arizona, four times as man

notorcylces were registered in 1978 as in [966, while over five times as many notorcycle fatalities occurred in 1978 as n 1966. Motorcycles constitute ıpproximatley 4% of all registered motor ,ehicles in the State while, by contrast, notorcycle fatalities represent over 8% of ıll motor vehicle fatalities. The Arizona Department of Transportation's accident ;ummary for 1977 reports that over nine notorcyclists are injured per day in Arizona and that one motorcyclist is killed about every five days. Further statistics describing motorcycle accidents n Arizona in 1977 and 1978 include the following:

1. Over half of all fatal motorcycle ıccidents in the years 1977 and 1978 occurred between the hours of 6:00 p.m. ınd 1:00 a.m. The worst days of the week for motorcycle accidents were Friday and Saturday.

2. The two most common circumstances that contributed to fatal motorcycle accidents were: a) speed too fast for conditions, b) had been drinking (this constitutes *by far*; the greatest contributing circumstance, being a factor in at least 26% of all motorcycle fatalities in 1978).

3. Most accidents and fatalities occurred on local streets, as opposed to controlled access routes or state/U.S. highways.

4. A particularly unhappy aspect of the motorcycle accident problem in Arizona is that almost 90% of drivers invovled in accidents in 1978 were under age 35 and *40% of the drivers were under the age 21.*

How to Improve the Odds

Physicians and other health professionals can exert their influence to encourage motorcyclists to optimize their chances of a safe and enjoyable ride in the following ways:

1. Wear a helmet, not only to decrease the risk of death, but also to reduce the risk of incapacitating, and sometimes neurologically severe, chronic injuries. Full face helmets are best.

2. Avoid riding after 6:00 p.m., especially on weekends.

3. Always avoid alcoholic beverages prior to motorcycle riding. The risk of a fatal accident for a motorcycle rider who has been drinking is far greater than that for an automobile driver who has been drinking.

4. Ride not only safely but defensively, since many fatal accidents are caused by the "other guy." Be especially cautious at intersections.

5. Always ride with the motorcycle's *headlight on.* (This has been shown to *substantially* improve the ability of other motorists to see the motorcycle.).

6. Wear conspicuous clothing. An increase in the popularity of flashy, bright colored clothing similar to what is worn by skiers would be helpful.

On a community basis, we should vigorously offer our opinions to legislators when questions regarding safety and health hazards like this are raised. We should encourage motorcycle safety courses such as the Motorcycle Safety Foundation's Rider education Course. Perhaps we should demand that such a course be prerequisite to obtaining a motorcycle operator's license. Despite the difficulties involved, we should encourage stricter enforcement of the current helmet law which applies only to those under 18. If this were more strictly enforced, a possible benefit could be the discovery of those operators who do *not* have valid licenses. We need to have more research and development to find helmet designs that are more acceptable to motorcyclists, especially in the hot summer months here. Finally, I cannot help commenting on the need for automobile drivers to be more aware of and cautious around motorcycles.

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Special thanks to Ross E. Kelley, Safety Projects Services at the Arizona Department of Transportation, for providing data on Arizona accidents, and to Begtgs Nelson of the Gold Wing Road Riders Association for his assistance with this article.

Suggested additional reading: Some of the most fascinating and valuable research in motorcycle safety is being undertaken by the University of Southern California under the direction of Dr. Harry Hurt. Some of this results are quoted in the article. Dr. Hurt is himslef and avid motorcyclists. Reference #6 listed above is a report of his work.

ARIZONA ACCIDENTS IN 1977 FOR MOTORCYCLES COMPARED TO ALL VEHICLES

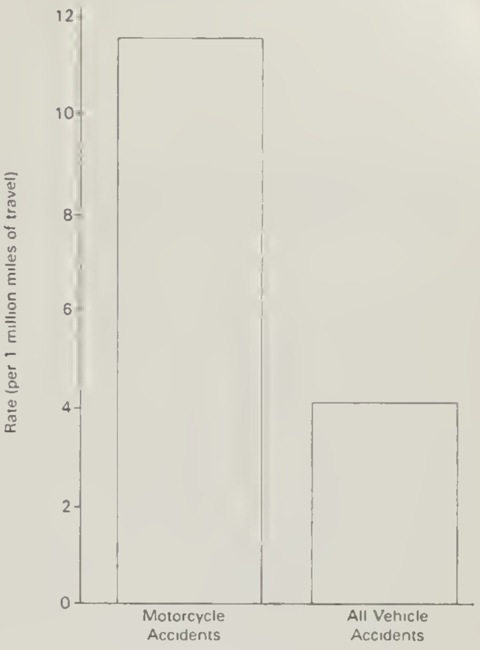

Figure 1

ARIZONA FATALITIES IN 1977 FOR MOTORCYCLES AS COMPARED TO ALL VEHICLES

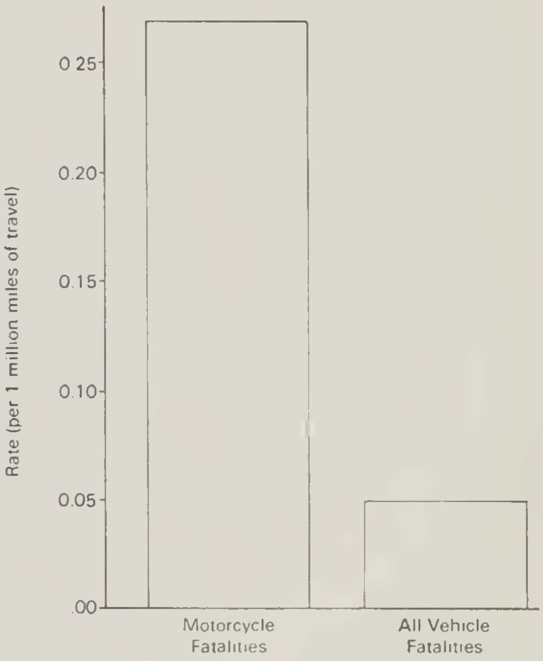

Figure 2

adiology Case of the Month

Continued from page 604

Spontaneous Gastric Perforation

The patient was taken to surgery and the discovery of a three (3) cm tear along the greater curvature of the stomach was made. A large amount of formula was aspirated from the peritoneal cavity. The infant recovered postoperatively without complication. No predisposing etiology could be identified.

Although spontaneous gastric perforation is an infrequent diagnosis, it is nevertheless the most common neonatal gastrointestinal perforation. Spontaneous rupture usually occurs in otherwise normal patients but there are certain predisposing factors, e.g., prematurity and neonatal asphyxia. The etiology is unknown but congenital gastric muscle deficiency, peptic ulceration, distal bowel obstruction, and iatrogenic causes (nasogastric tube erosion, excessive distension of the stomach from feeding, and vigorous resuscitation) have been implicated.

Clinically, these patients do well until the third to fifth day of life. Gastric perforation is hallmarked by the sudden development of abdominal distension, often following a feeding and is associated with emesis, irritability, and respiratory distress.

As in this case, the most common site of rupture occurs along the greater curvature of the stomach. Treatment is surgical and consists of suturing the perforation and lavage of the peritoneum to remove gastric contents from the abdominal cavity.

Radiographically, a massive pneumoperitoneum is present. A large intraperitoneal air fluid level indicating spillage of gastric contents is often present. The is demonstrated best on upright or left lateral decubitus views (Figure B). On the supine radioggraph the pneumoperitoneum is characterized by the presence of 1) diminished density projected over the peritoneal cavity and 2) visualization of the falciform ligament as a diagonal linear soft tissue density. This combination of findings has been described as the "football sign" (Figure A). The outer wall of the bowel may also be seen, outlined by intraperitoneal air and intraluminal air. This is known as the bas relief or "double wall" sign (Figure B).

Other considerations in the differential diagnosis of neonatal pneumoperitoneum include necrotizing enterocolitis, small or large bowel obstruction (meconium ileus, intussusception, or bowel atresia), or perforated duodenal ulcer. Rupture of a volvulus, perforation of a Meckel's diverticulum, or iatrogenic perforation of the colon (i.e., rectal thermometer insertion) must also be considered. Pneumoperitoneum may also occur secondary to a respirator induced pneumomediastinum.

Gastric perforation can usually be differentiated from other causes of pneumoperitoneum by the clinical presentation and radiographic findings. A massive pneumoperitoneum, especially with a large intraperitoneal air fluid level, is most consistent with gastric perforation. Spontaneous rupture of the stomach has significant mortality (50%). The earliest possible recognition and treatment of the condition is necessary for optimal prognosis.

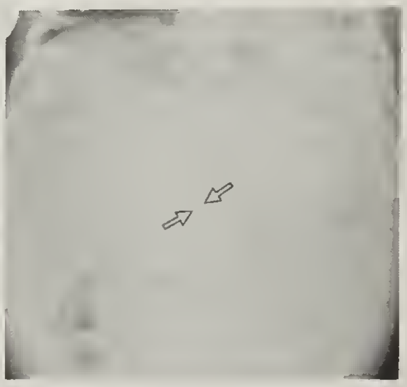

Figure A
Note the generalized decreased density throughout the peritoneal cavity. The falciform ligament is evident (arrows).

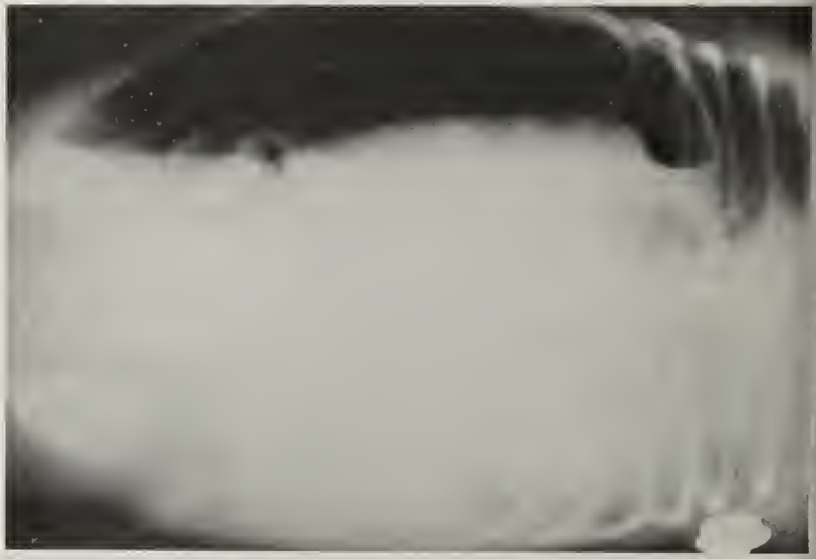

Figure B
Observe the massive pneumoperitoneum with an air fluid interface in the left lateral decubitus view.

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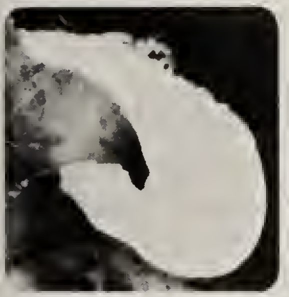
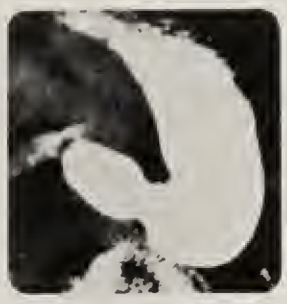


Pylorospasm has almost totally blocked passage of barium meal.



Barium meal beginning to pass 10 minutes after intramuscular injection of 20 mg. Bentyl.

"The correlation of spasm relief and drug given was excellent."

*This drug has been classified "probably" effective in treating functional bowel/irritable bowel syndrome.

†See Warnings, Precautions and Adverse Reactions.

See following page for prescribing information.

Reference:
King, J.C. and Starkman, N.M.: Evaluation of an antispasmodic. Double-blind evaluation to control gastrointestinal spasms occurring during radiographic examination. A preliminary report. Western Med. 5:356-358, 1964.

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Brief Summary

INDICATIONS
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For use in the treatment of infant colic (syrup).

Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS: Obstructive uropathy (for example, bladder neck obstruction due to prostatic hypertrophy); obstructive disease of the gastrointestinal tract (as in achalasia, pyloroduodenal stenosis); paralytic ileus, intestinal atony of the elderly or debilitated patient, unstable cardiovascular status in acute hemorrhage, severe ulcerative colitis; toxic megacolon complicating ulcerative colitis; myasthenia gravis. WARNINGS: In the presence of a high environmental temperature, heat prostration can occur with drug use (fever and heat stroke due to decreased sweating). Diarrhea may be an early symptom of incomplete intestinal obstruction, especially in patients with ileostomy or colostomy. In this instance treatment with this drug would be inappropriate and possibly harmful. Bentyl may produce drowsiness or blurred vision. In this event, the patient should be warned not to engage in activities requiring mental alertness such as operating a motor vehicle or other machinery or perform hazardous work while taking this drug. PRECAUTIONS: Although studies have failed to demonstrate adverse effects of dicyclomine hydrochloride in glaucoma or in patients with prostatic hypertrophy, it should be prescribed with caution in patients known to have or suspected of having glaucoma or prostatic hypertrophy. Use with caution in patients with. Autonomic neuropathy. Hepatic or renal disease. Ulcerative colitis. Large doses may suppress intestinal motility to the point of producing a paralytic ileus and the use of this drug may precipitate or aggravate the serious complication of toxic megacolon. Hyperthyroidism, coronary heart disease, congestive heart failure, cardiac arrhythmias, and hypertension. Hiatal hernia associated with reflux esophagitis since anticholinergic drugs may aggravate this condition.

Do not rely on the use of the drug in the presence of complication of biliary tract disease. Investigate any tachycardia before giving anticholinergic (atropine-like) drugs since they may increase the heart rate. With overdosage, a curare-like action may occur. ADVERSE REACTIONS: Anticholinergics/antispasmodics produce certain effects which may be physiologic or toxic depending upon the individual patient's response. The physician must delineate these. Adverse reactions may include xerostomia; urinary hesitancy and retention; blurred vision and tachycardia, palpitations; mydriasis; cycloplegia; increased ocular tension, loss of taste; headache; nervousness; drowsiness; weakness; dizziness; insomnia, nausea; vomiting; impotence; suppression of lactation; constipation, bloated feeling, severe allergic reaction or drug idiosyncrasies including anaphylaxis; urticaria and other dermal manifestations; some degree of mental confusion and/or excitement, especially in elderly persons; and decreased sweating. With the injectable form there may be a temporary sensation of lightheadedness and occasionally local irritation. DOSAGE AND ADMINISTRATION: Dosage must be adjusted to individual patient's needs.

Usual Dosage. Bentyl 10 mg. capsule and syrup: *Adults:* 1 or 2 capsules or teaspoonfuls syrup three or four times daily. *Children.* 1 capsule or teaspoonful syrup three or four times daily. *Infants:* ½ teaspoonful syrup three or four times daily. (May be diluted with equal volume of water.) Bentyl 20 mg.: *Adults:* 1 tablet three or four times daily. Bentyl Injection: *Adults:* 2 ml. (20 mg.) every four to six hours intramuscularly only. NOT FOR INTRAVENOUS USE. MANAGEMENT OF OVERDOSE: The signs and symptoms of overdose are headache, nausea, vomiting, blurred vision, dilated pupils, hot, dry skin, dizziness, dryness of the mouth, difficulty in swallowing, CNS stimulation. Treatment should consist of gastric lavage, emetics, and activated charcoal. Barbiturates may be used either orally or intramuscularly for sedation but they should not be used if Bentyl with Phenobarbital has been ingested. If indicated, parenteral cholinergic agents such as Urecholine® (bethanecol chloride USP) should be used.

Product Information as of October, 1978

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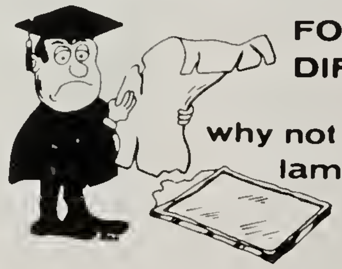

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State of the Art

HDL Cholesterol - Fact and Controversy

Lawrence P. Temkin, M.D.

Eric H. Prosnitz, M.D., Editor

The association of abnormalities of blood lipid concentrations and the process of atherosclerosis has long been recognized. The laboratory measurement of total serum cholesterol and triglyceride levels has allowed the identification of individuals considered to be prone to the development of coronary artery disease and has led to a practice of dietary, physiologic, and pharmacologic intervention therapy in an attempt to alter risk. In addition to the role of cholesterol and triglyceride determination, recent emphasis has been directed to the measurement of serum lipoproteins, complex particles necessary for the hematogenous transport of both dietary and endogenously produced lipids to sites of peripheral utilization and storage. It has been suggested that the binding of cholesterol to one specific lipoprotein species, high density lipoprotein (HDL), represents a potential mechanism for elimination of lipid from sites of peripheral deposition. This discussion will attempt to review current concepts of the function of serum HDL in cholesterol homeostasis and the evidence and hypotheses suggesting its potential protective role in the genesis of coronary artery disease.

Lipoprotein Classification, Characteristics and Management

The laboratory methods generally available for the determination of total serum cholesterol and triglyceride levels do not allow measurement of the relative concentrations of individual lipoproteins. The direct separation and quantification of distinct lipoprotein moieties can be accomplished by techniques based on difference in particle density (ultracentrifugation), difference in particle charge (electrophoresis), or difference in particle size (gel chromatography). The characteristics of the four major lipoprotein classes identifiable in serum are shown in Table 1. The most widely accepted nomenclature is that based on ultracentrifugation. This naming system, based on difference in particle density, will be utilized in this review.

Chylomicra, the major vehicle for serum transport of intestinally absorbed dietary lipid, are absent in fasting sera and have not been associated with atherogenic potential or cardiac risk. These particles are essentially devoid of cholesterol and composed almost completely of triglyceride.

Very low density lipoproteins (VLDL) are synthesized by the liver as the result of the catabolism of carbohydrate and free fatty acid. This lipoprotein fraction represents the major mechanism for transport of triglyceride from the liver to peripheral tissue for utilization. Cholesterol content of these particles by mass is small. At the peripheral tissue site, the particles are acted upon by the

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From: University of Arizona Health Sciences Center, Dept. of Internal Medicine, Section of Cardiology, Tucson, AZ 85724. Dr. Temkin is Assistant Professor of Medicine and Director, Cardiac Catheterization Laboratory.

Table 1
Serum Lipoprotein Classification

Ultracentrifuge Class	Electrophoretic Class	Lipid Content (% of mass)	
		Triglyceride	Cholesterol
chylomicra	—	85	5
very low density lipoprotein (VLDL)	pre-beta	60	15
low density lipoproteins (LDL)	beta	10	45
high density lipoproteins (HDL)	alpha	5	15

enzyme lipoprotein lipase leading to the release of glycerol and free fatty acid.

The remnant of VLDL catabolism by lipoprotein lipase, now rich in cholesterol after release of triglyceride, is termed *low density lipoprotein (LDL).* It has been hypothesized that this moiety serves as a source of cholesterol for peripheral tissue membrane synthesis.[1] It has been shown that specific LDL receptors exist at the peripheral cell membrane where these particles selectively bind. The particles are then internalized into the cell by endocytosis and the lipoprotein degraded. The cholesterol ester component is hydrolyzed and the resultant free cholesterol either is incorporated into the cell membrane or re-esterified and accumulated intracellulary. Peripheral tissue other than skin, liver, or adrenal cortex cannot catabolize cholesterol. As such, a mechanism to allow elimination of cholesterol from sites of tissue deposition has been hypothesized. This metabolic function is thought to be aided by *high density lipoprotein (HDL).*[2]

Plasma HDL are thought to be produced by liver and intestinal mucosa. In addition, a mechanism for HDL production from remnants of chylomicra has been postulated. The particle contains 90% protein by weight. A major phospholipid component of the particle is lecithin. In the periphery, the enzyme lecithin cholesterol acyltransferase (LCAT) catalyzes the reaction: lecithin + cholesterol → cholesterol ester + lysolecithin. This reaction additionally requires the presence of an apoprotein of HDL. The lysolecithin product is removed by binding to plasma albumin. The esterified cholesterol product derived from free cholesterql is incorporated into the matrix of the HDL particle in a plasma soluble form. A gradient is thereby established for further cholesterol to enter the particle and theoretically to be removed from cellular deposition sites. When all lecithin has been converted, the particle is saturated with esterified cholesterol. Therefore, it would appear that the major portion of circulating HDL particle is composed of the end products of the LCAT reaction.[3] In general, HDL carry approximately 25% of the circulating cholesterol content in serum.

The fate of the HDL particle cholesterol content i unclear. It has been postulated that HDL may provide transport mechanism for cholesterol back to the liver o serve as a medium for transfer of cholesterol to large lipoprotein particles. Levels of HDL are inversel correlated to isotopically determined total bod cholesterol stores.[4] It appears that this lipoprotein clas is essential for the control of the cholesterol pool an may limit peripheral tissue cholesterol storage. Indeed the clinical state of absence of HDL, Tangier disease, i associated with marked tissue cholesterol deposition particularly in skin and reticuloendothelial tissue.[5]

The direct quantitation of individual lipoprotei fractions by techniques previously mentioned is bot expensive and time-consuming and therefore no routinely applicable to clinical laboratory practice. A technique for measurement of HDL cholesterol leve (rather than HDL particle concentration) has bee developed based on the ability of heparin and divalen cation (Mn++ or Mg++) to precipitate LDL and VLD from solution in plasma.[6] Measurement of cholestero content in the supernatant fraction after precipitatio represents cholesterol affixed to HDL particles. Th error inherent in this technique may be as high as ± 10%

Levels of LDL similarly cannot easily be directl measured. Estimation of LDL cholesterol level can b made based on the measurement of total cholesterol HDL cholesterol, and total triglyceride using th formula: LDL cholesterol = (total cholesterol) - (HDL cholesterol) -(total triglyceride/5).[7] This estimate i accurate in fasting samples in the absence of chylomicr if triglyceride levels are less than 400 mg/dl.

HDL cholesterol levels in the normal population var according to sex, race, and age.[8] In general, norma population HDL cholesterol level is 50˙ 14 mg/dl. Level in women are approximately 10 mg/dl higher than i men and levels for blacks are approximately 10 mg/d higher than for whites. Concentrations are lowest a birth, but are fairly constant in adulthood.

Lipoproteins and Coronary Artery Disease

The association of elevated levels of total seru cholesterol and clinical manifestations of coronar artery disease was first recognized approximately 4 years ago.[9] Multiple population studies have confirme the relationship of clinically apparent coronary diseas and increased serum cholesterol level. In addition, th potential predictive value of serum cholestero measurement for subsequent development of clinica coronary artery disease has been recognized. Severa other important factors have become apparent. Th results of longitudinal study in the Framingham coho questioned the concept of a "normal" range fo laboratory measurement of total cholesterol.[10] Relativ risk for symptomatic coronary disease was found t correlate to the serum cholesterol value at any leve within the range of measurement. In patients below 5 years of age subsequently developing coronary hear

Table 2
Relative Risk for Coronary Artery Disease
Based on Total Cholesterol/HDL Cholesterol Ratio

Risk	Total Cholesterol/HDL Cholesterol Men	Women
0.5 x average	3.43	3.27
average	4.97	4.44
2 x average	9.55	7.05
3 x average	23.99	11.04

Table 3
Relative Risk of Coronary Artery Disease
Based on LDL Cholesterol/HDL Cholesterol Ratio

Risk	LDL Cholesterol/HDL Cholesterol Men	Women
0.5 x average	1.00	1.47
average	3.55	3.22
2 x average	6.25	5.03
3 x average	7.99	6.14

disease, average serum cholesterol was 244 mg/dl, well within most laboratory "normal" ranges. In addition, at any level of total cholesterol, risk was augmented by concomitant cigarette smoking, hypertension, or glucose intolerance. The majority of coronary events occurred in individuals with only mild to moderate elevations of serum cholesterol level, thus limiting the value of the measurement as an independent predictor for the individual patient.

The ability to separate distinct lipoprotein serum components, as previously described, has allowed additional insight into the risks implied by the total cholesterol measurement. Elevated LDL cholesterol measurements have been strongly correlated to enhanced cardiac risk in all ages examined. Levels of VLDL, predominantly triglyceride in content, have not proven to be predictive. At present, triglycerides are not considered to be atherogenic.[10]

The role of HDL in the cholesterol-atherogenesis relationship was first commented upon in 1951 with the recognition of low alpha lipoprotein (HDL) levels in patients with manifest atherosclerosis.[11] Additional studies in Finland suggested the association of low HDL levels with disease occurred even though total serum cholesterol levels were within a "normal" range.[12]

As the biochemical transport role of HDL in cholesterol homeostasis became more fully understood, the potential function of HDL in retarding the development of atherosclerosis was speculated upon by Miller and Miller in 1975.[13] Prospective population analysis in the Framingham group indicated that HDL cholesterol level at the time of entrance into the study was the single most sensitive individual predictor for the development of coronary event in patients 49-82 years of age. The lower the level of HDL, the greater the risk.[14] HDL cholesterol level was more highly predictive than LDL cholesterol. Similar results were noted for patients between age 20 and 49 in the Tromso cohort in Norway.[15] An additional group of families were identified with constitutionally elevated levels of HDL that appeared to be associated with increased longevity.[16]

Angiographic confirmation of the association between HDL cholesterol level and coronary disease has recently appeared.[17] In addition, an inverse correlation of the HDL cholesterol measurement and the severity of vessel involvement in angiographically identified coronary artery disease has been suggested.[18]

HDL Cholesterol and Assessment of Coronary Risk

It becomes clear from the preceding discussion that prospective population study has indicated that measurement of individual lipoprotein levels offers an enhanced predictive capability for the subsequent development of coronary disease as compared to the measurement of total cholesterol alone. Clearly, at any level of total cholesterol concentration, multiple groups can be identified based on the percentage of the total represented by cholesterol bound to HDL. To account for this relative HDL contribution, the calculation of the ratio [total cholesterol/HDL cholesterol] has been recommended as a risk predictor. Table 2 represents risk stratification data from the Framingham study based on this calculation. For example, a male individual with a total cholesterol of 250 mg/dl, within the "normal" range, and an HDL cholesterol level of 25 mg/dl, would have a calculated ratio of 10.00 yielding a relative predicted risk for coronary heart disease somewhat greater than twice average. Another individual with the same total cholesterol level and an HDL cholesterol of 65 mg/dl would yield a ratio of 3.85, indicating a less than average risk.

Even greater predictive capability has been claimed if, in addition to HDL cholesterol, the contribution of LDL cholesterol determined either by direct measurement or by estimation as previously described is taken into account.

The risk data based on the calculation of [LDL cholesterol/HDL cholesterol] ratio is shown in Table 3. It is assumed that this calculation balances the potential deleterious effect of the LDL contribution against the potential beneficial effect of the HDL fraction. Clearly, the higher the ratio, the greater the risk of coronary artery disease.

It should be noted that the risk data presented here are based on lipid analysis alone. At any level of lipoprotein concentration, an individual risk in excess of that listed is established by the associated presence of cigarette smoking, hypertension or glucose intolerance.

Factors Affecting HDL Cholesterol Level

Levels of HDL cholesterol appear to be dependent upon environmental factors in addition to genetic predisposition.

Cigarette Smoking. It has been noted in the Framingham study that smokers have lower HDL levels than non-smokers and that this reduction is not correlated with the duration of smoking, but rather to the daily intensity of consumption.[19] It has been suggested that HDL levels return to normal within one year of cessation of smoking.

Obesity, Diet and Weight Loss. Several studies have confirmed a negative correlation between weight and HDL cholesterol levels when populations are examined. Individuals with higher weights generally show lower HDL cholesterol measurements.[14] [20] Prospective dietary control of total cholesterol and saturated fat consumption in the Oslo cohort has suggested increases of approximately 20% in HDL cholesterol level after four years in a selected group of highly responsive hypercholesterolemic male patients between the age of 40-49.[21] Similar beneficial effects of dietary control to lesser degrees have been reported in other groups studied. The relative contribution of dietary modification as opposed to weight loss induced by the diet is difficult to ascertain from the data available.

Ethanol. Alcohol consumption has been demonstrated to be associated with a diminished prospective risk for myocardial infarction.[22] HDL cholesterol levels approximately 10% above those of non-drinkers have been demonstrated in groups consuming an average of 5-6 ounces of alcohol per week, the equivalent of approximately one cocktail per day.[23]

Physical Training. It has been observed that HDL cholesterol levels are highest in marathon runners and elevated in joggers as compared to sedentary individuals[24] suggesting a potentially beneficial role for physical training. Prospective data recently presented in the setting of physicial conditioning with maintenance of body weight at pretraining levels revealed decrease in total cholesterol with insignificant decrease in HDL cholesterol at the completion of the program.[25] It would appear that the higher HDL cholesterol levels noted in trained individuals need not be as a result of physical conditioning per se, but rather associated with dietary and weight control accompanying the runner's lifestyle.

Control of Hypertension. Little change in serum HDL levels has been associated with administration of antihypertensive medication. Recently, however, it has been noted that groups receiving propranolol therapy manifest diminished HDL levels as compared to groups receiving other forms of therapy.[26]

Patient Management

The demonstration of the potential for modification of diet, cigarette and alcohol consumption, and physical activity to increase HDL cholesterol levels has led to the speculation that risk for development of coronary disease might be favorably affected by appropriate intervention. It must be noted, however, that the magnitude of change demonstrated for laboratory measurement of HDL cholesterol levels in many of these studies was no more than 4-5 mg/dl, changes potentially falling within the range of technical error of the measurement as previously mentioned.

In the absence of improved methodology, if one is to assume the small changes noted to be truly significant, is there justification to warrant intervention in individua patient management directed specifically at increasing HDL cholesterol levels? Prospective experience in the Multiple Risk Factor Intervention Trial (MRFIT) demonstrated small but probably insignificant change in group HDL levels as a result of simultaneous control of smoking, diet and hypertension.[27] Within the group studied, large individual variation was noted, however with several individual increases of as much as 10 mg/d in level. No data has been presented to date to suggest a direct effect of intervention on HDL level to decreased risk for the development of atherosclerosis.

It appears from the information presented that what would be considered routine risk factor management may not consistently increase serum HDL cholestero levels. It should be noted, however, that with the possible exception of propranolol administration, this form of combined therapy probably does not adversely affect the concentration of this lipoprotein.

It would seem reasonable at present to include lipoprotein profiling and HDL cholesterol measurement as part of routine risk factor identification screening in individuals with low to moderate levels of tota cholesterol, even though these values may be within the range of a laboratory norm. Patients identified with low HDL cholesterol levels with high risk profile ratios might potentially benefit from risk factor management therapy, although this cannot be proven.

The risk for development of coronary artery disease must be considered to be multifactorial and specific therapy oriented at modification of HDL level exclusively does not appear to be warranted at present. Control of hypertension, cessation of cigarette consumption, and dietary modification are all therapies where the potential risk and cost appear to be minimal, while the potential benefits cannot yet be assessed. As such, cardiac risk factor modification directed at these major components would appear to be indicated and the associated effects on HDL cholesterol would appear to be of secondary consequence and concern. The results of additional prospective studies now in progress must be awaited before specific HDL-directed therapies can be recommended.

Conclusion

Assessment of serum HDL cholesterol level has allowed enhanced capability for evaluation of potential risk for the development of coronary artery disease. The value of alteration of HDL cholesterol level for potential amelioration of risk has yet to be demonstrated.

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Arizona
Medicine

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Editorials

What is a Successful Editorial

Writing editorials are difficult. It is essential to have a topic that is of interest to a large portion of the audience and the topic must be important enough to warrant the allotted space. An editorial should also have a purpose, i.e., to praise, to point out difficulties or to stimulate thought. The latter criterion is the hardest to fulfill for what is purposeful to one may be misinterpreted by someone else and thus prove to be controversial. The last series of editorials on "Training of Internists" produced responses ranging from condemnation to praise. The fact that the editorial aroused any response is significant. The fact that the conflicting viewpoints were so vocal and came from well-intentioned and concerned physicians made the editorial stimulating. But was that enough? I think not. In order to be successful an editorial in its role of pointing out deficiencies or stimulating thought needs to have its readership respond. As yet, no written communication about these editorials has been received. In order to resolve a problem there has to be discourse. I'm hopeful that this written silence will not reflect the actions of those involved in the training of internists in Arizona. An editorial cannot be labeled "successful" until time has lapsed so that a perspective of its impact can be appreciated.

Marshall B. Block, M.D.
Editor

Why Additonal Space at the College of Medicine?

Recent studies by the College's Long Range Planning Committee, an ad hoc committee composed of basic scientists, and several in-depth academic departmental reviews have identified a need for additional College of Medicine space for teaching and research. This need is the result of a number of factors. First of all, the College was designed and built for a maximum class size of 64 medical students in each year. Each class now has at least 88 students.

Secondly, some College departments have changed their research and service emphasis in ways that were not originally anticipated. This accounts for new space requirements in the Department of Radiology, for example. This department was conceived as a service/clinical research unit, but recent directions in cancer and other research have moved the Department toward instrument design (Computerized Axial Tomography [CAT] for example), imaging (better non-invasive means of detecting disease) and "bench" research for studying the effects of radiation on cells. These changes in teaching emphasis and investigation require larger amounts of different types of space than originally planned.

Thirdly, some of our present departments and division did not exist at the time the College was designed. Anesthesiology and Neurology were sections in traditional departments but now are separate departments. The Cancer Center Division and the division of Respiratory Sciences did not exist. Their presence as separate units ecognizes the importance of their eaching role in the College of Medicine. For units which did not result simply rom administrative rearrangement, pace is required so faculty may develop and express research goals from which new knowledge may be made available to esidents of the state of Arizona.

A fourth reason for the increased need or space is the complex type of reserach onducted these days in university settings. Instrumentation for most esearch is large and one often needs everal instruments even for the most imited investigative approaches. Although clinical research at the bedside continues to be an important part of the generation of new knowledge, more and more a biomolecular approach to disease is needed. So called "wet laboratories" to support such clinical research require more space than that used in descriptive empirical or "bedside" research. In-depth backup investigation at the bench is required in modern clinical drug testing trials, in research on drug toxicology, metabolism, cancer therapy, and antibiotic research.

As a prompt and expedient solution to the College's space needs, 25,000 square feet of research space has been developed in modular temporary buildings adjacent to the Health Sciences Center. Funding for this construction was secured from outside gifts and donations and from the clinical faculty through its Medical Service Plan. The construction has been rapid and at a modest cost. These buildings will relieve the immediate space shortage. Although temporary, the space allows the Health Sciences Center to continue to develop, leaving time for considering realistic planning for permanent contruction.

Based on changes in education, health care delivery, population and health needs of the state, planning continues at the College of Medicine. Funds from private sources, government, and generated income will be used to accomplish the changes needed to stay current with the space needs prompted by the changes. While considering these everchanging influences upon us, we hope to retain flexibility, use temporary solutions where appropriate and plan wisely any permanent expansion necessary to meet the health education

Louis J. Kettel, M.D.
Dean

Change - The Reality, The Challenge

Heraclitus - "There is nothing permanent except change"

Our profession is undergoing dramatic changes that are having and will have profound effect for physicians and our patients, unless we, too, make changes as individuals and as a group.

Peer review and hard work have long been accepted as part of our profession. In the past, this was balanced by the respect and gratitutde of our patients along with a comfortable lifestyle. The demands of consumerism and malpractice claims, the rip-tide of increasing government regulation, the control of our diagnostic and therapeutic facilities by hospital administrators and other entrepreneurs, our dependence on the whims of the third parties, and the threat of the loss of the right to be self-employed, have the physician nervously reacting to these divisive forces that are impersonal to his efforts and beyond his control.

The resultant frustration has the doctor lashing out at organized medicine, whom he feels doesn't represent him in the way he sees the teamsters represented. We want the teamster clout, but we stay aloof from the battle and splinter our only unified voice. Organized medicine is you and I - if we don't like it, change it - get involved, but don't abandon the only hope we have at this critical time.

The great question that confronts us in medicine is this: Can our profession, which is always more concerned with the quality of medical care of our patients than the cost, provide the leadership to develop the necessary changes in health care to satisfy a concerned public who already faces the ravages of double digit inflation.

The spiraling health care costs are a concern to all citizens, doctors included. Medicine is a victim of its own success. We have attained a remarkable level of technical achievement such as renal dialysis, coronary by-pass, joint replacement, etc. but this advanced technology gives highly specific medical benefits for a select group of clinical problems which afflicts only a small segment of the population. This same technology, however, has created public expectation beyond economic reality.

Many inceased costs are part of the generalized inflation. A pernicious increase in health care costs has been precipitated by unwarranted hospital capital expenditures and unnecessary reduplication of medical facilities and equipment. Doctors are held responsible for these costs by the public.

Unfortunately, physicians have also contributed to this cost because we exert a profound influence on spending. We want many of these new facilities in our hospital, and we also hospitalize the patient (which represents 70% of the health care dollar.)

f Medical expenditures and regulation by the federal government have contributed immeasurably to the inflated costs of health care. Government regulations add as much as $45.00 per day of hospitalization. Every regulation robs us of some of our freedom - our right to decide.

The public's aspirations and expectations for good health are insatiable. In a society where the standard of living continues to improve, the rate of conversion of wants to needs becomes ever more rapid. The general public equates health with a right, and expects it to be dispensed by our profession. We have conditioned the public to expect a "penicillin cure" for evey illness, and a pill for every emotion.

We in medicine have accepted the political axiom that everyone is entitled to medical care, but failed to educate the public that they, individually, are primarily responsible for their health. There is a difference between health and medical care. Health is not a function of new discoveries, new machines, or new beds. Health is relative to individual habits. Although health care costs are 8.9% of the gross national product - the public spends 5% of the gross national product on alcohol and tobacco. These two items alone are responsible for 35% of health care costs.

In addition, we have failed to educate the people and the politicians that illness is also the end product of many of society's problems, such as pollution, housing, a sedentary existence, and environmental stress.

Medicine has also taken the criticism for costs without informing the public of the benefits they received. There are now

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Presidential address presented at the 88th Annual Meeting of the Arizona Medical Association, Wednesday, April 25, 1979.

ewer hospital days per illness. More people than ever before survive major illnesses and are completely rehabilitated to a full and longer life. The cost should be equated to the benefits received.

Our aging population requries more care for broad spectrum of chronic debilitating disease. The averaage annual expenditure for medical care from 19 - 65 years is $547.00, from 65 on its $1,521.00.

All these discrepancies and complexities in our medical system are currently in evidence, but they are not understood or appreciated by the public. Depsite the fact that the recent polls by Gallup and the Johnson Foundation have shown that 86% of the population in all economic divisions of society are satisfied with their medical care, fully 53% of the population including a majority of physicians, think that national health insurance is needed to control costs and increase access. Although this dicotomy of thought is mystifying, this is what we must deal with -this is the "real world."

The doctor is intersted in quality care for the individual patient. The politician professes a wider responsibility of adequate care for the health and welfare of the people within the restraints of the resources available. The politician believes that expanded medical care can be delivered within strict economic restraints, maintaining the expected quality, and still give unlimited accessibility! This is not possible. Such rhetoric is geared toward re-election, not reality.

National health insurance is not the answer. There are no expamples in which government intrusion into the private sector has resulted in a less expensive, better quality product. We do not fear government involvement - it is government incompetence. Regulation has proven to be cumbersome, stultifying, expensive and inefficient.

Quality medical care cannot be assured by making the doctors observe a set of government -initiated "proforma" reguatlion. At best, adhearence to such regualations promotes mediocrity. Above all, bureaucratic regulation of medical practice does not provide compassionate and caring medicine. *Government cannot do anything for you that it hasn't already done to you!* Government can force compliance but *not* commitment.

Doctors must reconstruct the medical system to preserve the competition of private enterprise along with the valued doctor-patient relationship.

To do this we can no longer remain under the cloak of self-serving professionalism. No longer can professionalism deal only with our relationships with our patients, our relations with each other, or our approach to the science and art of medical practice. From now on professionalism must also deal with the economics of our work - our ability to deliver quality, cost-effective care, patient education, and physician leadership necessary to implement the changes, to preserve what we discern as valuable and necessary to the quality of medical care, and to eradicate what is not cost-effective.

The answer to our problems, as put by Shakespeare, "The fault, dear Brutus, lies not in the stars, but in ourselves."

Our profession - medicine, is a mixture of philosophy, science, morality, and magic, dealing with the variables of the individual. It too, however, has a common denominator -economics - who pays the bill. No longer does the patient pay the bill directly. Third parties - industry and government - via insurance companies have been interposed between the doctor and his patient.

Health insurance companies have only given lip service and a few billboards to cost containment. There interest lies in keeping their precentage of the growing premium for as long as they can, just as they did with our malpractice insurance. The insurance industry has compiled a dismal record in hosptial reimbursement, and they intend to proselyte the entire field of health care if left unchallenged.

The only way to really reduce medical cost is to make our population healthier. Improved health of our people is as big a challenge as trying to hold down the cost of care that results from poor health.

Human nautre is the mjaor obstacle to preventive medicine. Most of our health problems are caused by our lack of discipline - smoking, drinking too much, eating too much, not using seatbelts, and lack of effective exercise. The pattern of our dialy lives heavily influences our health and how long we live.

People value good health and 92.5% of Americans recognize their personal responsibility for health, and feel that changing their lifestyle would do more to improve their health than anything the doctors and medicine could do. This is what they think - *not* what they do.

The medical profession and hospitals, as well as business, labor, and government must agree to divert more than the present 3% of our total medical bill to preventive health measures.

Doctors and hospitals are trained to manage illness and are paid for this service. To interest the medical system in better heatlh, there has to be an economic incentive for everyone.

The incentive for business is a lower medical bill, less absenteeism, better production. Labor can see healthier workers who can negotiate for broader benefits to enhance health rather than cover illness.

What is the incentive to doctors? The doctors should reassert their leadership by designing, owning, and operating real health centers to which the person at risk can be sent to improve their health. 20% of the working population accounts for 80% of the costs of medical care. These people can be identified by genetics, their lifestyle, and the survey tests that we now have. Once identified, these same individuals can be encouraged to go to our health centers with third party payment, for the behavioral changes that will obviate or reduce the risk of serious illness. Why should the more disciplined, healthy person pay for the people who do not take personal responsibility for their own health? Individuals who fail to make the necessary behavioral change for better health should pay a larger share of their own health insurance. Personal health care can be put on an economic incentive basis.

Doctors and responsible hospitals should establish health care alliances that would provide comprehensive care in return for a capitation premium.

Doctor-owned individual practice association (I.P.A's) and hospitals would then be at risk together to provide alternatives that would minimize government intrusion into the health care system. Hospital diagnostic and therapeutic departments that are now used only 8 hours a day could be used 16 hours a day by allowing the doctor - owned I.P.A. to lease these facilities during their unused hours for outpatient use. Such effective utilization of capital equipment would reduce consumer costs.

Physicians must work together to fashion cost-effective mangement of various medical problems that would be interdigitated with a preventive system. Outpatient medicine should be the province of the physician and the economic incentive would be directed to keeping people out of the hospital.

Conservative estimates indicate that such programs should reduce hospitalization by 35%.

The competition of competing systems would encourage hospitals to be innovative rather than monpolistic purveyors of health services under the guise that they are non-profit.

We must also encourage society to be more involved in the ethical decisions of our profession. Should a chronically ill 80-year-old have a cardiac pacemaker or a heroic cancer operation? Should everyone have the benefit of complete, total care not matter what the prognosis and the anticipated cost?

As physicians we have a moral obligation to be invovled with society in solving these dilemmas.

A knowledgeable citizenry is the basis for mutual understanding between the public and the medical profession, and will ultimately result in imaginative and responsible public policy.

The profession must not become satisfied or defensive with what exists, and deny the need for change. If we prefer local peer regulation to bureacratic domination, we must make these chages to maintain control of our profession and the direction of organized medicine.

We have all enjoyed the benefits of the effort and sacrafice of those that have preceeded us in our profession - we too must dedicate ourselves, for the physicians of the future. Doctors can no longer tamely follow - we must control our destiny. This is our responsibility, this is our trust.

Change is difficult - but it is the difficult things in life that make us grow.

With new incentivies, and doctor-hospital cooperation, we *will* make the necessary innovative changes that will preserve the things in medicine that make t the most highly valued profession.

William E. Crisp, M.D.
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In Memoriam

Jerome Gerendasy, M.D.

1917 - 1979

J. W. Kennedy, M.D.
History and Obituaries Editor

Arizona lost a good friend and a gifted physician with the death of Jerome Gerendasy on January 30, 1979.

Dr. Gerendasy was a unique human being. Although he started this life in moderate surroundings in New York City, he went on to graduate from New York University, then studied medicine at St. Andrews in Endinburgh, Scotland, and graduated from the Lausanne Medical School in Switzerland.

Perhaps because of this diversified background. Dr. Gerendasy developed an enormous compassion for people from all walks of life. He was able to sense the needs of each individual and relate to people at all levels, both in an intellectual and emotional sense. The highest testimonial is that his patients regarded him as a friend as well as a physician.

Dr. Gerendasy's career in internal medicine and cardiology spanned only 37 years, but the measure of the man cannot be taken in years, but rather in accomplishments. And his were legend. . .

*Three years as a Major in the U.S. Army, European Theatre.

*Director of Cardiology at the Hospital for Joint Diseases and Medical Center and at Madison Avenue Hospital, New York.

*Associate Physician at Flower Fifth Avenue Hospital, Metropolitan Hospital and Bird S. Coler Hospital in New York.

*Chairman of the Department of Medicine at Desert Samaritan Hospital in Mesa, Arizona

*Diplomate of the American Board of Internal Medicine.

*Fellow of the American College of Physicians, Cardiologists and Chest Physicians.

*Fellow of the New York Academy of Medicine.

*President of the New York Cardiology Society.

But to dwell on Dr. Gerenday's medical accomplishments would not do full justice to the man, for there were so many varied facets to his life: music. . .photography. .

horticulture. . .linguistics. . .world travel. . .politics. . .literature. . .history, ad infinitum. He was so impressively knowledgeable in all of these areas of man, that one might almost have considered him to be imposing had it not been for his delightful sense of humor which always belittled his own accomplishments and praised those of others.

His greatest pride, however, was in his wife Vera and daughter Lynn, both of whom he loved dearly.

As his partner, I know that his patients loved him greatly and that all of us, his family, associates, patients and friends, will miss his dedication to medicine and the enrichment he gave to all of our lives.

Edward A. Smith, M.D.

Lloyd K. Swasey, M.D.

1904 - 1979

Lloyd was born in Tonopah, Nevada on April 4, 1904. He and his brother Paul (killed in an aeroplane accident) were early on thrown on their own resources. Following graduation from Tonopah High School, Lloyd worked his way through pharmacy school at the University of Southern California. He returned to Tonopah and for a few years was content with pharmacy as a profession. His chief interest was medicine and was accepted at the University of Colorado as one of the "older" students. He continued incidental employment during his medical school days in order to pay expenses, graduating from the University of Colorado in 1935. He thereafter served his intership at Fresno County Hospital in Fresno, California. His brother Paul enticed him to come to Phoenix for a residency at Good Samaritan Hospital. Before entering private practice he took a postgraduate course at New York University where he became interested in pulmonary disease. He returned to Phoenix and entered practice with Dr. Eugene Gatterdam and Dr. Jesse Hamer. For many years his office was maintained in the Professional Building at 15 East Monroe Street. His years in Phoenix covered 1938 to 1975 with a "leave" of four years to serve in the United States Navy. His terminal assignment was in Hawaii and following his discharged from the Navy as a Commander he returned to Phoenix to practice. For many years he was the mainstay of the Arizona Tuberculosis Sanatorium in Tempe, Arizona. He worked diligently for the welfare of the patients afflicted with tuberculosis and his efforts were rewarded by the establishment of the new Arizona Tuberculosis Sanatorium. He was appointed Medical Director of the new sanatorium and he continued in that capacity from 1963 through 1973 when the sanatorium was closed. He served on the Board of Directors of the National Tuberculosis Association for ten years and was a well respected specialist in pulmonary disease.

Following the untimely death of his first wife Mary Lou, he was later married to Norma Lee Goff on November 1, 1957. From this union there was born Lloyd Kenneth Swasy, Jr., and Susan Lynn. His daughter by his first marriage, Cynthia Anne Schreyer blessed him with three grand-children; Robert, Sherri and Kathryn. Norma and the children were quite solicitous of Lloyd and during the terminal phase of his incapacity they were guardian angels in a most difficult situation.

The state of Arizona and Lloyd's colleagues are indebted to him as a shining example of a dedicated, sincere physician. He practiced the time honored mode of doing as good as possible today with hopes for a brighter tomorrow. We will miss him and our heartfelt sympathy goes out to Mrs. Swasey and the children.

Dermont W. Melick, M.D.

ArMA Reports

THE MINUTES APPEARING IN THIS SECTION HAVE BEEN EDITED TO CONSERVE SPACE. A COMPLETE COPY OF THE MINUTES OF ANY MEETING WILL BE MAILED TO ANY MEMBER REQUESTING THEM.

Executive Committee

The meeting of the Executiv Committee of the Arizona Medica Association, Inc. held at 810 Wes Bethany Home Road, Phoenix, Arizona on Thursday, June 7, 1979, a quoru being present, convened at 6:15 p.m William E. Crisp, M.D., President an Chairman, presiding.

Mica Proposal Re: Contract and Committees

The committee received for its revie the action of the MICA Board of Trustee taken May 22, 1979, the report of MICA' Subcommittee on Review Committee and the current guidelines for the Clai and Underwriting Review Committees. Drs. Myers, Sattenspiel and Morac MICA's Subcommittee on Revie Committees, addressed the committe relative to the bases for their findings an recommendations. Following a length discussion, Dr. Crisp appointed committee, consisting of James D. Alwa Jr., M.D., Richard L. Dexter, M.D Chairman, Neopito L. Robles, M.D. an Neil O Ward, M.D., to review, with th aid of counsel for ArMA and MICA, th recommendations of MICA's subco mitteee as they related to both t contract with MICA and the guidelin for the Calim and Underwriting Revie Committees. It was agreed tha subsequent to such review, this new a hoc committee would report back to th Executive Committee with it recommendations as to how ArM should proceed.

For purposes of fully informing th Committee, Drs. Alway and Ward at th point interjected their thought regarding new proposed guidlines f their respective committees, all of whic were to be incorporated into th determinations of the newly-apppointe committee, which will make every effo to timely meet and formulat recommendations for consideration b the Executive Committee in the ne future.

Economic Organization For Arizona Physicians

The committee considered the actio taken by the Board of Directors on Apr 25, 1979, relative to the formation of a economic organization as a part of the Arizona Medical Association for the purpose of negotiating contracts, carrying on insurance grievances for its members and monitoring third parties and determined that the committee would present to the Board of Directors at its next meeting five physicians who have indicated willingness and ability to not only act as such an economic organization but to participate in such negotiating seminars as may be available o more fully inform and prepare them or the task to be undertaken.

Several names were considered and it was the consensus that these physicians be contacted for an expression of interest n participating in such an organization. Dr. Crisp is to develop this suggested list.

Membership Program

The committee was apprised of AMA's recent offer to help state associations in heir ongoing solicitation for new members by way of sending someone to review and evaluate our present membership program and procedures and returning to ArMA with recommendations as to additional ways by which poteintial members might be approached. At this point, Dr. Crisp appointed John T. Clymer, M.D. as Chairman of the Ad Hoc Membership Committee, which ommittee, it was agreed, should avail tself of the AMA offer.

Insurance Review Committee

Requests of the Health Insurance Association of America, Metropolitan Life Insurance Company, Aetna Life & Casualty, Pacific Mutual Life Insurance Company and the Equitable Life Assurance Society of the United States elative to the establishment of a tatewide Peer Review Committee. RECEIVED.

Opinion letter from legal counsel dated May 15, 1979. RECEIVED.

Following lengthy disucssion regarding he items received for information.

IT WAS MOVED AND CARRIED THAT THE HEALTH INSURANCE ASSOCIATION OF AMERICA, METROPOLITAN LIFE INSURANCE COMPNAY, AETNA LIFE & CASUALTY, PACIFIC MUTUAL LIFE NSURANCE COMPANY AND THE EQUITABLE LIFE ASSURANCE SOCIETY OF THE UNITED STATES BE ADVISED THAT THE ARIZONA MEDICAL ASSOCIATION COULD NOT AID OR PARTICIPATE IN THE ESTABLISHMENT OF A STATEWIDE NSURANCE REVIEW COMMITTEE.

AMA Jail Proposal

The committee reviewed a recently ssued Summary of American Medical Association Program to Improve Medical Care and Health Services in Jails and, while the committee felt the program was worthy one, it did not feel that the inancial burden should fall upon the state association, and

IT WAS MOVED AND CARRIED TO REFER THE AMA JAIL PROPOSAL TO THE PROFESSIONAL COMMITTEE FOR ITS USE IN CONTACTING THE LOCAL OFFICIALS FOR THE LAW ENFORCEMENT ASSISTANCE ADMINISTRATION IN DETERMINING WHETHER THEY WERE, IN FACT, DESIROUS OF SUCH A PROPOSAL, WHETHER STATE FUNDS COULD BE MADE AVAILABLE AND WHETHER ANY FURTHER ACTION BY THE ARIZONA MEDICAL ASSOCIATION IN THISE REGARD WAS INDICATED.

Housestaff & AMA Convention

The request of the Housestaff of ArMA for ArMA's funding of two resident delegates to the AMA 1979 Annual Meeting in Chicago was considered by the committee.

IT WAS MOVED AND CARRIED THAT THE ARIZONA MEDICAL ASSOCIATION PROVIDE FUNDS FOR TWO DELEGATES FROM ARMA'S HOUSSESTAFF SECTION TO ATTEND THE JULY 1979 ANNUAL MEETING OF THE AMERICAN MEDICAL ASSOCIATION IN CHICAGO.

Western Physicians Purchasing Association

John F. Kahle, M.D., President of WPPA, spoke to the committee regarding membership status of the WPPA, of which Arizona has provided 221 memberships, consisting of 383 physicians, and sought the committee's assistance insofar as immediate solicitation of more members. It was suggested that each member of the Board of Directors be contacted by letter requesting that they speak with the physicians in their area who have joined WPPA and then, on a one-to-one basis contact those physicians who have not, attempting to make them more fully aware of the advantages of the new ArMA benefit. Dr. Kahle additionaly commented on new product line being added and the opinion letter from the Federal Trade Commission.

Correspondence

Commendation letter for Gary L. Barnett from the Mutual Insurance Company of Arizona dated 5/2/79. RECEIVED.

OTHER BUSINESS

Medical Economics Committee - Industrial Commission of Arizona

A discussion was held regarding the recent ICA hearing and a draft letter from the Association to the Industrial Commission of Arizona was presented to the committee for its review and determination.

IT WAS MOVED AND CARRIED THAT THE LETTER PRESENTED TO THIS COMMITTEE BE PREPARED, WITH MINOR CHANGES, IN FINAL FORM FOR SUBMISSION TO THE INDUSTRIAL COMMISSION OF ARIZONA AND THAT THE LETTER BE MADE A PART OF THESE MINUTES.

MICA Insurance Department Report.

Mr. Robinson addressed the committee relative to audits by the Insurance Department of the Mutual Insurance Company.

IT WAS MOVED AND CARRIED TAHT BRUCE E. ROBINSON, AS EXECUTIVE DIRECTOR OF THE ARIZONA EMDICAL ASSOCIATION, BE AUTHORIZED ON BEHALF OF THE ASSOCIATION TO DO ANY AND ALL THINGS NECESSARY TO COMPLY WITH THE FINDINGS OF THE INSURANCE DEPARTMENT AS THEY RELATE TO ITS AUDIT OF MUTUAL INSURANCE COMPANY OF ARIZONA.

Annual Meeting Change

Mr. Robinson advised the committee that, because of a breach by The Registry Resort of its contract with the Association for the holding of the 1980 meeting at its facility, the Scientific Assembly Committee had determined to look elsewhere for a possible location and that, although the dates of the meeting must be changed, so that it will be one week earlier than planned, i.e., April 23-27, 1980. The Pointe Resort has now been contracted with for the holding of the 1980 Annual Meeting of the Association.

IT WAS MOVED AND CARRIED TO APPROVE THE POINTE RESORT AND APRIL 23-27, 1980 AS THE NEW LOCATION AND DATES FOR THE 1980 ANNUAL MEETING, AS REQUESTED BY THE SCIENTIFIC ASSEMBLY COMMITTEE.

Funding Requests

The committee was presented with a request for funding from the Governor's Council of Arizona Women for Highway Safety to aid in the printing and distribution of brochures on the necessary use of highway restraints for children and from the Pima Council to Immunize Arizona's Children to aid in the printing and distribution of immunization records. Following a discussion of both matters,

IT WAS MOVED AND CARRIED THAT, WHILE BOTH THE PROJECT OF THE GOVERNOR'S COUNCIL OF ARIOZNA WOMEN FOR HIGHWAY SAFETY AND THE PROJECT OF THE PIMA COUNCIL TO IMMUNIZE ARIZONA'S CHILDREN ARE FELT TO BE TREMENDOUSLY IMPORTANT, DESERVING AND WORTHWHILE, NO FUNDS ARE PRESENTLY AVAILABLE FOR THE ASSOCIATION TO GIVE FINANCIAL SUPPORT AT THIS TIME, BUT THAT BOTH REQUESTS WOULD BE HELD IN ABEYANCE TO BE RECONSIDERED AS CIRCUMSTANCES ALLOWED.

GOVERNMENTAL SERVICES COMMITTEE

Meeting of the Governmental Services Committee of the Arizona Medical Association, Inc., held Wednesday, June 20, 1979, at 810 W. Bethany Home Road, Phoenix, convened at 6:52 p.m., Louis C. Kossuth, M.D., Chairman, presiding.

Welcome

Doctors Davis, Peters, and St. John were warmly welcomed as new members to the Committee.

The Chairman welcomed Mrs. John Clymer and thanked her for attending this meeting. He informed the Committee of the Auxiliary's plans to monitor HSA meetings in the future. This function is in the earliest planning stages, but a great deal of enthusiasm is being generated.

Chairman's Report

Project Review Manual

The Chairman informed the Committee that the "Project Review Manual" has been approved by the SHCC. Several lists of projects coming up for review in the near future were received by the members. It was noted that in the development of this manual, cost containment and effectiveness were examined and incorporated into the criteria.

Medical Advisory Committee

The Committee was informed that we have received a response to our offer of a medical advisory committee to the SHCC. We have been requested to submit the name of the designated committee chairman.

Annual Meeting Report

It was noted that Resolution 2--79, P.L. 93-641; Composition of HSA Boards of Directors and Resolution 3-79, National Health Insurance, presented to the House of Delegates by the Governmental Services Committee at the Annual Meeting in April were adopted as amended.

AMA Midwest Regional Health Planning Workshop

Dr. Kossuth attended the AMA Midwest Regional Health Planning Workshop held in Chicago in June 15-16, 1979. His report was received by the Committee and is on file at the ArMA offices. One point that was repeatedly made was that the health planning guidelines are only guidelines and can be adjusted to meet local problems.

Program for the Coming Year

The Chairman requested the members to submit ideas for subjects for future meetings of the Governmental Services Committee. Some of the suggestions for topics for future meetings were: Food Additives; Nuclear Power Regulation (Radiation); News Releases Relative to Medicine (It was suggested that AMA be contacted to see if there is any way that government agencies, i.e. HEW, FTC, VA, can supply the state medical associations a copy of their medicine related news releases directly.); and Elimination of Restrictions on Nominations to CAHSA.

Understanding Health Planning

A report entitled "Understanding Health Planning" written by Dr. Kossuth was received by the Committee. This report is on file at the ArMA offices. Using the report as a guideline, discussion ensued on the subjects of the Annual Implementation Plan, Project Review, Appropriateness Review and Development Grants. This was a lively discussion in which it became evident that not enough physicians are involved in the health systems agencies and further that physicians in general do not completely understand what the health systems agencies are, what they do and how physician input can help. It was apparent during the discusssion that physician input is vital to proper and informed health planning. There was considerable discussion on the possibility of a workshop on health planning (P.L. 93-642) for physicians and especially the Association's Board of Directors, Committee Chairmen, County Society Officers and appropriate Auxiliary members.

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE BOARD OF DIRECTORS THAT THE ARIZONA MEDICAL ASSOCIATON SPONSOR A WORKSHOP ON THE GENERAL SUBJECT OF HEALTH PLANNING (P.L. 93-641), HOW IT WORKS IN ARIZONA AND HOW IT AFFECTS PHYSICIANS.

Future Medical Meetings

CONTINUING MEDICAL EDUCATION

The following institutions and organizations have been accredited for their continuing medical education programs by the Liaison Committee on Continuing Medical Education.

Arizona Heart Institute
Arizona Medical Association, Inc.
Arizona State Hospital, Phoenix
Camelback Hospital, Phoenix
Desert Samaritan Hospital, Mesa
Good Samaritan Hospital, Phoenix
Health Maintenance Associates, Phoenix
Maricopa County General Hospital, Phoenix
Mesa Lutheran Hospital, Mesa
Phoenix Baptist Hospital & Health Center
Phoenix Indian Medical Center
Phoenix Memorial Hospital
Phoenix Psychiatric Council
St. Joseph's Hospital & Medical Center, Phoenix
Barrow Neurological Institute, Phoenix
St. Lukes's Hospital & Medical Center, Phoenix
Scottsdale Memorial Hospital
Tucson Hospitals Medical Education Program, Tucson
Veterans Administration Hospital, Phoenix
Veterans Administration Hospital, Prescott
University of Arizona College of Medicine, Tucson

The accredited Institutions and organizations above produce a variety of continuing medical education programs. Each accredited Institution and organization is responsible for designating which of these programs meet ArMA's requirements for Category 1 credit. Physicians who participate in programs which are designated Category 1 by accredited Institutions will receive Category 1 credit toward the ArMA Certificate in CME and the AMA's Physician's Recognition Award.

AUGUST

ACLS Instructor Course

August 17 & 18, 1979. Arizona Medical Assoc Office, 810 W. Bethany Home Rd., Phoenix AZ 85013. Sponsor: Arizona Affiliate of the American Heart Assoc. Contact: Caro Turner, ArMA Offices, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 16 hours category I credit.

Rocky Mountain Cancer Conference

August 17-19, 1979. Broadmoor Hotel, Colorado Springs, Co. Sponsor: American Cancer Society Colorado Medical Society & Colorado Society of Osteopathic Physicians. Contact; Rocky Mountain Cancer Conference. 1809 E. 18th Ave., Denver, Co. 80218.

NEOSPORIN® Ointment

(Polymyxin B-Bacitracin-Neomycin)

Wellcome

Burroughs Wellcome Co.
Research Triangle Park
North Carolina 27709

Each gram contains: Aerosporin® (Polymyxin B Sulfate) 5,000 units, bacitracin zinc 400 units, neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base), special white petrolatum qs; in tubes of 1 oz and 1/2 oz and 1/32 oz (approx.) foil packets.

INDICATIONS: *Therapeutically,* (as an adjunct to systemic therapy when indicated), for topical infections, primary or secondary, due to susceptible organisms, as in: infected burns, skin grafts, surgical incisions, otitis externa; primary pyodermas (impetigo, ecthyma, sycosis vulgaris, paronychia); secondarily infected dermatoses (eczema, herpes, and seborrheic dermatitis); traumatic lesions, inflamed or suppurating as a result of bacterial infection. *Prophylactically,* the ointment may be used to prevent bacterial contamination in burns, skin grafts, incisions, and other clean lesions. For abrasions, minor cuts and wounds accidentally incurred, its use may prevent the development of infection and permit wound healing.

CONTRAINDICATIONS: This product is contraindicated in those individuals who have shown hypersensitivity to any of its components. Do not use in the eyes or in the external ear canal if the eardrum is perforated.

WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

SEPTEMBER

ACLS Recertification Course
September 21, 1979. Arizona Medical Association offices, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Sponsor: Arizona Affiliate of the American Heart Assoc. Contact: Carol Turner, ArMA offices, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

OCTOBER

Second Annual Symposium on Lifestyles and Health.
October 13-14, 1979. Sheraton-Place Hotel, San Francisco. Sponsor: Univ. of California San Francisco Schools of Medicine, Nursing, Denistry and Pharmacy. Contact: Continuing Education Health Sciences, 24 Kirkham, San Francisco, CA 94143. CME credit available.

ACLS PROVIDER COURSE
October 19 & 20, 1979. Arizona Medical Associaiton offices, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Sponsor: Arizona Affiliate of the American Heart Assoc. Contact: Carol Turner, ArMA Offices, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

Eighth Annual Symposium of the Western Division of the American Geriatrics Society. Management of Vascular Disease in the Elderly
October 26-27, 1979. Lakes Club, Sun City Arizona. Sponsor: University of Arizona College of Medicine and Walter O. Boswell Memorial Hospital. Contact: Julius Schwimmer, M.D., 13220 105th Ave. Bldg. B, Rm. 61, Sun City, AZ 85351, Approved for 11 hours of category 1 credit toward ArMA, AMA, & AAFP CME Certificates

NOVEMBER

Symposium on Stress
November 3 & 4, 1979. Sheraton-Palace Hotel, San Francisco, Ca. Sponsor: University of California Schools of Medicine, Nursing, Denistry, and Pharmacy. Contact: Univ. of California, Continuing Education Helath Sciences, 24 Kirkham, San Francisco, CA 94143. CME Credit available.

Southern Medical Association Annual Scientific Assembly
Las Vegas, Nevada, November 4-7, 1979, Sponsor: Southern Medical Assoc., & AMA. Contact: AMA Dept. of Meeting Services, 535 N. Dearborn St., Chicago, ILL. or Southern Medical Assoc., 2601 Highland Avenue, Birmingham. Alabama 35205.

27th Annual Meeting of the Medical Society of the United States and Mexico
November 14-17, 1979. Doubletree Inn, Tucson, Az. Sponsor: Medical Society of the United States and Mexico. Contact: Mrs. Carolyn Parsons, Executive Secretary, 3161 N. Pantano Road, Tucson, AZ 85715.

Arizona Society of Otolaryngology and Maxillo-Facial Surgery.
November 16-18, 1979. The Wigwam, Litchfield Park, AZ. Sponsor: Arizona Society of Otolaryngology & Maxillo-Facial Surgery. Contact: Neil O. Ward, M.D., 4550 N. 51st Ave., Phoenix, AZ 85031.

The Third Annual National Symposium on Aging
November 17 & 18, 1979. Holiday Inn, Golden Gateway, San Francisco, CA. Sponsor: Univ. of California San Francisco. Contact: Univ. of California, Continuin Education Health Sciences, 24 Kirkham, San Francisco, Ca 94143.

1980

FEBRUARY

Second Annual International Cataract Surgery Sumposium
February 18-20, 1980. Sponsor: Arizona Fundation for Ophthalmic Research and Education. Contact: Ronald W. Barnet, M.D., Medical Director, 421 N. 18th St., Ste 105, Phoenix, AZ 85006. (602) 257-1077. Approved for 18 hours of category 1 credit.

Pediatric Update 1980
February 25-28, 1980. Sponsor: St. Joseph's Hospital and Medical Center, Dept. of Pediatrics. Contact: Ronald A. Christensen, M.D., Dept. of Pediatric Education, St. Joseph's Hospital and Medical Center, 350 W. Thomas Rd., Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

Contmeporary Management of Stroke an Coronary Disease.
February 27-March 1, 1980. Lodges in Taos New Mexico. Sponsor: Symposia de Santa Fe. Contact: W. J. Levy, M.D., Symposia de Santa Fe, P.O. Box 5175, Santa Fe, Nev Mexico 87502.

MONTHLY OR WEEKLY

Shrine Medics Meeting
Second Tuesday of each month, Doctor Hospital, 5:45 p.m. J. South Classroom Sponsor: Shrine Medics. Contact: Robert C Briggs, M.D., 5121 N. Central Ave., Phoeni AZ 85012.

Office Psychiatry for the Primary Provider
2nd Monday of month, 4811 N. 7th Stree Phoenix, AZ. Sponsor: Arizona Health Pla Contact: T.R. Bittker, M.D., Box 500 Phoenix, AZ 85010. Approved for category credit.

Counter Transference Group
Weekly, Thurs. 8—10 p.m. Sponsor: Phoeni Psychiatric Council. Contact: James E Campbell, M.D., 5051 N. 34th St., Phoenix, A 85018. Approved for 2 hours category credit.

Monthly Meeting of Tucson Radiologists
Last Tues. of month. Plaza Internationa Tucson, AZ. Sponsor: U of A Medical Cente Dept. of Radiology. Contact: Irwin M Freundlich, M.D., U of A Health Science Center, Dept. of, Radiology, Tucson, A 85724. Approved for 2 hours category credit.

Pediatric Grand Rounds
Tuesday 7:30-8:30 a.m in Phoenix: 1s Tues.—Phoenix Indian Hospital, 2nd Tues.— Maricopa County Hospital, 3rd Tues.—Goo Samaritan Hospital, 4th Tues.—St. Joseph' Hospital. Sponsor: Maricopa Count Hospital (Phoenix Hospital's Affiliate Pediatric Program). Contact: J. Kip Charlton, M.D., 2601 E. Roosevelt, Phoenix AZ 85008. Approved for 1 hour per sessio category 1 credit.

Review of Forensic Pathology Current Case, Special Topics
Thursday, weekly, 11 a.m., 120 S. 6th Ave. Phoenix, AZ. Sponsor: Arizona Society o Pathologists. Contact: H.H. Karnitschnig M.D., 120 S. 6th Ave., Phoenix, AZ. Approve for 1 hour per session category 1 credit.

ARIZONA STATE HOSPITAL

2500 E. Van Buren, Phoenix, AZ. 85008
Contact: Arnold L. Kendall, M.D.

A.S.H. Psychiatric Grand Rounds
2nd Wed., 1:00-2:00 P.M., J-6 Conf. Rm., Contact: Dr. Conger & Staff

Clinical-Pathological Conference
3rd Wed., 1:30-2:30 P.M. General Services Bldg., Conf. Rm.

Medical Grand Rounds
4th Wed., 1:00-2:00 P.M., Medical Bldg. Conf. Rm.

Staff Education Conference
Weekly, Wed., 1 p.m., Contact: Howard E. Wulsin, M.D.

BARROW NEUROLOGICAL INSTITUTE

Medical Education
Barrow Neurological Institute of St. Joseph's Hospital and Medical Center, 350 W. Thomas Rd., Phoenix, AZ 85013. Sponsor: St Joseph's Hospital & Medical Center. Contact: John R. Green, M.D. Approved for 1 hour category 1 credit.

Neurology Teaching Conference
Tuesdays, 8:30-9:30 a.m., Eighth Floor Conf. Room.

Neurosurgery Morbidity Conference
Wednesdays, 8:15-9:15 a.m., on first and third weeks, Eighth Floor Conference Room.

Neuro-ophthalmology Conference
Tuesdays, 7:30 a.m. in 8th floor neurology conference room.

Spinal Injury Conference
Wednesdays, 8:15-9:15 a.m., on second and ourth weeks, in Neuropathology Conf. Rm.—a multidisciplinary review of admission by neurosurgeons, orthopedists, and ehabilitation specialists

Neuropathology of Gross Specimens Conference
Thursday, 7:30-8:30 a.m. in the Morgue.

Neurology-Neurosurgical Clinical-Neuroradiological and Neuropathological Conference
Fridays, 8-9 a.m., First Floor Conference Rm.

Neuropathology or Neuroradiology Conferences
Friday, 9 a.m., Neuropathology in Neuropathology Conference Rm., Neuroradiology in First Floor Conference Rm.

Neurorehabilitation Conference
Tuesdays, noon, 8th Floor Conference Rm.

Neurosurgical Journal Club
Saturdays, 9-11 a.m. in Eighth Floor Conference Rm.

CAMELBACK HOSPITAL

5055 N. 34th St., Phoenix, AZ 85018. Sponsor: Camelback Hospital. Contact: Medical Director.

Patient Staffing Conference
Three times weekly.

DESERT SAMARITAN HOSPITAL

1400 South Dobson Road, Mesa, Arizona, Contact: L.A. Rosati, M.D. Approved for category 1 credit. **Clinical Conference - Dept. of Medicine**
Weekly Thurs., 12:30-1:30 P.M. Buffet Lunch, Grande Rm.

Medical Education Programs
Wed.,7:00 P.M. Desert Samaritan Hospital, Mesa.

Monthly Visiting Professor Programs
Tuesday 7:00 P.M., Buffett 6:00 P.M.

OB/GYN Medical Staff Conference
Weekly, Monday, 12:30-1:30 P.M. Grande Rm.

Surgical Morbidity & Mortality Conference
2nd Thurs., 7:00 A.M., Grande Rm.

GOOD SAMARITAN HOSPITAL

1033 East McDowell Rd., Phoenix, AZ Contact: Warren A. Hinchcliffe, M.D. Approved for category 1 credit.

Anatomical Path. & Cytology
Weekly, Mondays 4:00-5:00 P.M., S-4 Conf Rm.

Gynecological Section Conference
2nd Monday, 12:30-1:30 P.M., West Rm

Monday Noon Conference
Weekly, 12:00-1:00 P.M. R-2 Conf. Rm.

Mortality/Morbidity, Surgery Comm.
2nd Monday, 11:30-2:00 P.M., West Rm.

Obstetrical Section Conference
1st Monday, 12:30-1:30 P.M., West Rm.

Pulmonary Disese Grand Rounds
Weekly, 12:00-1:00 P.M., D-5 North

Family Practice Conference
Weekly Tuesday, 12:00-1:00 P.M., Fam. Practice Conf. Rm.

Pediatric Grand Rounds
3rd. Tuesday, 7:300-8:30 A.M., West Rm.

Peri-Natal Mortality Conference
3rd Tuesday, West Rm. (May-August).

Surgical Grand Rounds
Weekly, Tuesday 7:00-8:30 A.M. East Rm.

Clinical Cancer Fourm
3rd Wednesday, 12:00-1:00 P.M., West Rm, Contact: John A. Bruner, M.D.

Combined Cardiovascular & Family Practice Conf.
Ist, 2nd, & 4th Wed., 12:00-1:00 P.M., D-5 North.

Dr. Finger's Lecture
Weekly, Wed., 12:30-1:30 P.M., R-2 Conf. Rm.

Learning Disabilities
Weekly, 10:30-12:30 A.M., R-2 Conf. Rm.

OBGYN Dept. Review & Statistics
Weekly, 12:30-3:00 P.M., OBGYN Conf. Rm.

PM&R Grand Rounds
1st. & 3rd. Wed., 12:00-1:00 P.M., R-2 Conf. Rm.

Gynecological Pathology
Weekly Thursday, 1:00-2:00 P.M., S-4 Conf. Rm.

GI Radiology Conference
1st Thursday, 12:00-1:00 P.M., F151, Radiology Conf. Rm.

Neurology Grand Rounds
Weekly Thursday, 9:00-10:00 A.M. D-5 North.

Tumor Treatment Planning Conf.
2nd & 4th Thursdays, 12:00-1:00 P.M., F151 Radiology

Joint Tumor Gyn Conference
2nd Friday, 12:30-1:30 P.M., West Rm.

Kinesology
Weekly Friday, 4:00-5:00 P.M., R-2 Conf Rm.

Medical Grand Rounds
Weekly Friday, 8:00-9:00 A.M., West Rm.

Neuropathy Conference
Last Firday, 1:15-2:15 P.M., S-4 Morgue.

Psychiatry Grand Rounds
Weekly Friday, 11:30-1:00 P.M. , East Rm.

Pediatric Wednesday Seminar
July, Sept., March 12:30-1:30 P.M., East RM.

Pediatric Thursday Seminar
May, June, July, 12:30-1:30 P.M., East Rm.

Phoenix Ultrasound Society Meeting
Sept., thru May,2nd Thursday, 7-9:00 P.M., Med. Ed. Bldg. Contact: Dr. A. Ben-Ora.

PM&R Journal Club
Alt. Fridays, 12:00-1:00 P.M., R-2 Conf. Rm.

Family Practice Grand Rounds
1st. Wed.: April, Aug. & Dec., 7:30-9:00 A.M., West Rm.

MARYVALE SAMARITAN HOSPITAL

Continuing Medical Education Program
Every other Wednesday, 7-8 a.m.

KINO COMMUNITY HOSPITAL

2800 E. Ajo Way, Tucson, AZ 85713. Contact: Eric G. Ramsay, M.D. Approved for category 1 credit.

Attending's Hour
5th Monday, 12:30 P.M., Contact: Joseph Goldberger, M.D.

Infectious Disese Conference
3rd Monday, 12:30 P.M., Contact: Larry Lincoln, M.D.

Nephrology Conference
1st Monday, 12:30 P.M., contact: Alan Cohn, M.D.

OB/GYN Dept. Meeting
Weekly Mondays, 7:30 A.M., Contact: Lewis Shenker, M.D.

Psychiatric Conference
2nd & 4th Mondays, Contact: Jose Santiago, M.D.

Surgical Conference
Weekly Mondays, Contact: Ronald Fischer, M.D.

Cardiology Conference
4th Tuesday, 12:30 P.M., Contact: Brendan Phibbs, M.D.

Gynecology Conference
5th Tuesday, 12:30 P.M., Contact Lewis Shenker, M.D.

Neurology Conference
1st & 3rd. Tuesday, 12:30 P.M., Contact: H.W. Buschsbaum, M.D.

Radiology Conference
2nd Tuesday, 12:30 P.M., Contact: Marvin Baker, M.D.

Clinical Pharmacology
4th Wed., 12:30 P.M., Contact: Lee Gardner, Pharm.D.
Clinical Pathology Conf.
2nd Wed., 12:30 P.M., Contact: Philip Altman, M.D.
Gynecology Pathology Conference
Weekly Wed., 7:3'45 A.M., Contact: Lewis Shenker, M.D.
Medical Conference-Mortality & Morbidity
3rd Wed., 12:30 P.M., Contact: Joseph Goldberger, M.D.
Rheumatology Conference
1st Wed. 12;30 P.M., Contact: George Werner, M.D.
Cardiology Conference
1st Thursday, 12:30 P.M., Contact: Brendan Phibbs, M.D.
Clinical Correlation
3rd Thursday, 12:30 P.M., Contact: Elliot Munjack, M.D.
G-I Conference
4th Thursday, Contact: Ray Moldow, M.D.
OB/GYN Sonography/X-ray Conference
Weekly Thursday, 9:15 A.M., Contact: Lewis Shenker, M.D.
Chest Conference
2nd & 4th Fridays, 12:30 P.M., Contact: David Engelsberg, M.D.
Dermatology Conference
3rd Friday, 12:30 P.M., Contact: Peter Lynch, M.D.
Hematology Conference
1st Friday, 12:30 P.M., Contact: Steven Ketchel, M.D.

MARICOPA COUNTY GENERAL HOSPITAL

2601 E. Roosevelt, Phoenix, Az 85008. Contact: Steven Schaffner, M.D.

Medicine Conference
Daily 12:00-1:00 P.M., Contact: Steven Schaffner, M.D.
Chest Surgery Conference
Weekly Mondays, 1:30 P.M., Satan Cruz Rm.
Obstetrical Problem Conference
Weekly Tuesday, 7:15 A.M., Yavapai Rm
Orthopedic Conference
Weekly Tuesday, 7:30 A.M., Santa Cruz Rm.
Pediatric Grand Rounds
2nd Tuesday, 7:30-8:30 A.M.
Hand Surgery Conference
Weekly Wed., 7:30 A.M., Santa Cruz Rm.
Neurosurgery Discharge Planning
Weekly Wed., 1:30 P.M., Station 42.
OB/Neonatology Seminar
Weekly Wed. 7:15 A.M., Yavapai Room
Psychiatry Conference
Weekly Wed. 11:00-12:00 A.M.
Surgery Conference
Weekly Wed., 7:00-8:00 A.M.
Cardiac/Cardiology Conference
Weekly Thurs. 12:00 P.M., Yavapai Room.
OB/GYN Resident Conference
Weekly Thurs. 12:00 P.M., Yavapai Rm.

GYN, Endocrine Seminar
Weekly Friday, 12:30 P.M., Santa Cruz Rm.
GYN Grand Rounds
Weekly Friday, 1:30 P.M., Santa Cruz Rm.
OB/GYN Surgical Pathology Conf.
Weekly Friday, 7:00 A.M., Yavapai Rm.
Orthopedic X-Ray Conference
Weekly Friday, 7:30 A.M., Santa Cruz Rm.

PHOENIX BAPTIST HOSPITAL & MED. CENTER

6025 N. 20th Ave., Phoenix, AZ 85015. Contact: James L. Grobe, M.D. Approved for category 1 credit.

Medical & Surgical Conference
Weekly Monday, 12;00 Noon
Medical Conference
1st. & 3rd Tuesdays, 12:00 Noon.
Medical & Surgical Conference
Weekly Wed., 12:00 Noon.
Medical & Surgical Conference
Weekly Friday, 12:00 Noon.

PHOENIX INDIAN MEDICAL CENTER

4212 North 16th St. Phoenix, AZ 85016. Contact: Leland L. Fairbanks, M.D. Approved for category I credit.

Pediatric Grand Rounds
1st Tuesday, 7:30-8:30 A.M.
Clinical Staff Teaching Conference (Family Practice)
Weekly Wed., 8:00-9:00 A.M., Conf. Rm. A.

PHOENIX MEMORIAL HOSPITAL

1201 S. 7th Ave., Phoenix, AZ 85036. Contact: George Scharf, M.D. Approved for Category 1 credit.

Monthly Medical Education Seminar
3rd Monday, 6:30 P.M., Kiva Conf. Rm.
Tumor Board Conference
Weekly Friday, 12:00 P.M., Kiva Rm., Contact: Howard Kimball, M.D.

SCOTTSDALE MEMORIAL HOSPITAL

7300 East 4th St., Scottsdale, AZ 85251. Contact: W. S. Williams, M.D. Approved for category 1 credit.

Family Practice Conference
1st Monday, 12:45 P.M.
Morbidity & Mortality Conference
2nd Monday, 12:45 P.M.
Medical Grand Rounds
3rd Monday, 12:45 P.M.
Neurology & Neurosurgery Conference
3rd Monday, 12:30 P.M., Doctors' Lounge
Internal Medicine-Clinical Pathlogical Conference
4th Monday, 12:45 P.M.
Pulmonary Medicine Conference
1st Tuesday, 12:30 P.M., Doctors' Lounge
Cardiology Conference
2nd Tuesday 12:30 P.M., Doctors' Lounge
Surgery Departmental Meeting
3rd Tuesday, 12:15 P.M., Doctors' Lounge
Thoracic & Vascular Surgery Conference
4th Tuesday, 12:30 P.M., Doctors' Lounge
Cardiopulmonary Conference (Didactic)
4th Tuesday, 12:30 P.M., Doctors' Lounge
Peri-Natal Mortality & Morbidity Conf.
2nd Wed., 12:30 P.M., Doctors' Lounge
Trauma Conference
3rd Thurs., 12:30 P.M., Doctors' Lounge
Tumor conference
Last Thursday, 12:30 P.M., Doctors' Loung
Psychiatry & Behavioral Science Conference
1st Friday, 12:30 P.M., Doctors' Lounge
GI Medical-Surgical-Radiologica Conference
2nd Friday, 12:30 P.M., Doctors' Lounge
Uronephrology-Oncology
Last Friday, 12:30 P.M., Doctors' Lounge

ST. JOSEPH'S HOSPITAL

350 West Thomas Road, Phoenix, AZ 8501
Contact: Joseph C. White, M.D.

OB/GYN Section Conference
3rd & 4th Mondays, 12:30-1:30 P.M., 1st Floo Conf. Rm.
Pediatric Grand Rounds
4th Tuesday, 7:30-8:30 A.M., Contact: J. Kip Charlton, M.D.

ST MARY'S HOSPITAL & HEALTH CENTER

1601 W. St. Mary's Road, Tucson, AZ 8570
Contact: see below.

Monthly Specialty Conference-Dept. o Surgery
1st Monday, 7:30 A.M., Century Rm. A Contact: Med. Staff Office.
Grand Rounds: Medical Surgical, Famil Practice, Pathology, Radiology
Weekly Thursday
Cardiology Conference
Weekly Friday, 8:00-9:00 A.M., Century rm Contact: Anthony Forte, M.D.

U OF A HEALTH SCIENCES CENTER

Sponsor: U of A College of Medicine, Tucson AZ 85724. Contact: See below. Approved fo category 1 credit.

Anesthesiology Lectures
Weekly Thursday, 4:00-5:00 P.M.
Cardiac Catheterization Conference
Weekly Friday, 4:00 P.M., Contact: Dr Temkin.
Cardiology Research Conference
Weekly Tuesday, 7:30 A.M., Contact: Dr Roeske.
Case Discussion
Weekly Wed., 7:00 A.M., Din. Rm.C&D., Contact: Drs. Vaughan & Doyle.
Clinical Immunology, Allergy & Rheumatology Rounds
Every Friday, Noon-1 p.m. Contact: John Boyer, M.D., Dept. of Internal Medicine.

1980
INTERNATIONAL CATARACT
SURGERY SYMPOSIUM
February 18-19-20, 1980

LaPosada Resort Hotel — Scottsdale, Arizona

FACULTY

Eric J. Arnott, England
D. Peter Choyce, England
Charles D. Kelman, United States
Edward L. Shaw, United States
Ronald W. Barnet, United States
Kenneth J. Hoffer, United States
Donald L. Praeger, United States
Harold Stein, Canada

For information please write:
Arizona Foundation for Ophthalmic
Research and Education
421 North 18th Street, Suite 105
Phoenix, Arizona 85006
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erebrovascular Disease Conference
ondays, 5-6 p.m., weekly, Rm. 5505. Contact: Wesly Moore, M.D.,Depts. of Surgery, Nurology & Radiology.

Dean's Clinical Rounds
Contact: Louis J. Kettel, M.D.

Dermatology Rounds
Weekly Wed., 11:00 A.M., Contact: Dr. Lynch

Ear, Nose & Throat
Alternate Wed., 4:00 P.M., Contact: Dr. Coulthard

Educaton Bloc
Weekly Thurs, 4:00-5:00 P.M., Rm 5403

Endocrinology Seminar
Weekly Thursday, 12:00-1:00 P.M., Contact: Dr. Nugent

Gastrointestinal Path. Conference
4th Friday, 1:30 P.M.

Head & Neck Tumor Meeting
Alternate Wed., 4:00 P.M., Contact: Dr. Manning

Hematology-Oncology Clinical Conference
Every Tuesday, Noon-1 p.m., 1st, 3rd & 5th Tuesdays, Rm 6505, Tucson Medical Center. 2nd & 4th Tuesdays, Rm. N318, VA Hospital. Contact: Sidney Salmon, M.D., Dept. of Internal Medicine.

Joint Thyroid Clinic Conference
1st & 3rd Mondays of every month.1 to 2 p.m., AHSC,Rm. 6505. Contact: Sidney Werner, M.D., Dept. of Internal Medicine, Endocrinology Section.

Nuclear Medicine
Thursday, 7:30 a.m., Radiology Conf. Rm., UAHSC. Contact: Irwin M. Freundlich, M.D., Dept. of Diagnostic Radiology.

Neurology Grand Rounds
Every Friday, Noon, 1st, 3rd, & 5th Fridays, UAHSC, Rm. 7505. 2nd & 4th Fridays, VA Hospital. Contact: William A. Sibley, M.D., Dept. of Neurology.

Neuromuscular Disese Conference
Weekly Friday, 11:00 A.M., Contact: Dr. Stern

Neuropathology Review Conference
Thursdays, 8:30 a.m., Rm. 5120, UAHSC. Contact: Peter C. Johnson, M.D.

Neuroradiology Conference
Weekly Thurs., 5:00 P.M., Contact: Dr. P.C. Christenson

Neurosciences Seminar
Every Friday, 7:30 a.m.,UAHSC, Rm 7505. Contact: Colin R. Bamford, M.D., or Jose F. Laguna, M.D., Dept. of Neurology.

Neuromus Journal Conference
2nd & 4th Thursday, 7:00-9:00 P.M., Contact: Dr. Stern.

Orthopedic Rounds
Saturday, 8:00 A.M., Contact: Dr. Peltier.

Pathology Seminar
Friday, 4:30-5:30 p.m., Rm. 5120, UAHSC. Contact: Paul Finley, M.D., Dept. of Pathology.

Pediatric Problem Patient Conference
Weekly Wed., 8:00 A.M., Contact Dr. Walson

Pediatric Research Forum
Weekly Tuesday, 7:30 A.M., Contact: Dr. James Jones

Pediatric Specialty Conference
Weekly Friday, 8:00 A.M., Contact: Dr. Philip Walson.

Psychiatric Grand Rounds
Weekly Wed., 12:00 Noon, Contact: Dr. Robert Cutts

(Psych.) Monthly Case Conference
2nd Friday, 7:30-9:00 A.M., Contact: Dr. Alan Levens

Pulmonary Rounds
Weekly Friday, 11:30 A.M., Contact Dr. Benjamin Burrowns.

Chest Radiology
Monday, 5-6 p.m., weekly, Rm 1535F, UAHSC. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Gastrointestinal Radiology
Mondays, 7:30 a.m., weekly except for summer months. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Radiation Oncology Planning Conference
Friday, weekly, 8:30-10 a.m., UAHSC, Rm. 0655. Contact: Robert S. Heusinkveld, M.D., Radiation Oncology Section.

Radiology-Rheumatology Conference
Thursday, 8 a.m., UAHSC, Library Rm.1535C. Contact: Irwin M. Freundlich, M.D. Dept. of Radiology.

Renal Pathology Conference
1st & 3rd Thurs., 11:30 A.M., Contact: Dr. Nagle.

Resident's Conference
Wednesday, weekly 5-6 p.m., Diag. Radiology Conf. Rm. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology

Uroradiology
Alternate Thurs, 5:00 P.M., Contact I.M. Freundlich

Surgical Literature/Basic Skills
Saturday, 8:00 A.M., Contact: Dr. P. Whitney

Surgical Morbidity & Mortality Conference
Weekly Wed., 8:00 A.M., Contact: Dr. Wangensteen.

Trauma Conference
Every Thursday,4-5 p.m. UAHSC, Rm. 5505. Contact: Martin Silverstein, M.D., Dept. of Surgery.

Grand Ward Rounds—Trauma
Every Tuesday, 8 a.m., UAHSC. Contact: Martin Silverstein, Surgery Dept., Trauma

Toxicology Conference
Weekly Tuesday, 8:00 A.M., Contact: Dr. Keith Likes
Section.

Monthly Meeting Tucson Ultrasonography Group
First Wed. of month, 4:30-5:30 p.m., UAHSC. Contact: Irwin M. Freundlich, M.D.

General Urology Conference
Weekly Thursday, 12:00 Noon, Contact: Dr. G. W. Drach.

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Cancer Symposium
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Cardiology Conference
2nd Thursday — 1 p.m. — T5, Dr
Habibzadeh, Ext. 348.

Clinicopathology Conference
4th Thursday, March, June, Sept. & Dec., 1:0
P.M., Contact Dr. Likos, Ext. 623

Dermatology Conference
1st, 2nd, & 3rd Wednesday (usually, but i
subject to change) 10 a.m. — C118, Dr
Roberts, Ext. 426.

G.I. Clinic
2nd & 4th Wed, 1:00-4:00 P.M.,

G.I. Radiology Clinical Correlation Conference 1st & 3rd Monday —
p.m. — C435, Dr. Sanowski, Ext. 618.

G.I. Grand Rounds
Weekly, Wed., 8:00 A.M., G-I Ward , Contact Dr. Severino

G.I. Resident Conference
Weekly Friday, 1:00 P.M., D446, Contacts Drs. Sanowski & Kozarek

G.I. Medical Surgical Conference
Weekly Friday, 2:00 P.M., Path. Conf. Rm.

G.I. Rounds
Weekly Thursday, 1:00 P.M., D446

Medical Service Grand Rounds
Weekly Friday — 11 a.m. — T5, Dr. Zeller, Ex
272

Medical-Surgical G.I. Conference
1st & 3rd Friday, 2:00 P.M., Rm. 2128, Contact: Drs. Kozarek and Mertz, Ext 493

Medical-Surgical Chest Conference
1st and 3rd Thursday — 1 p.m. — T5, Dr. Chowdhury, Ext. 388.

Surgery-Pathology Conference
Weekly Thursday, 2 p.m. — 2128, Drs. Mertz, Ext. 493 & Likos, Ext 623

Surgery Grand Rounds
Weekly Thursday, 3:00 P.M., Contact: Dr. Mertz, Ext. 493.

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Weekly Tuesday, 2:00 P.M., Contact: Dr. Young

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Weekly Wed., 12:00-1:00 P.M., V.A. Hospital Staff Conf. Rm. & (AHSC), Contact: Jay Smith, M.D.

Surgical Grand Rounds
Weekly Wed., 4:00 P.M., Contact: Dr. Putnam

Endocrinology Seminar
1st.,3rd, & 5th Thurdsays, 12:00-1:00 P.M., Rm. N318, Contact: Dept. of Internal Medicine.

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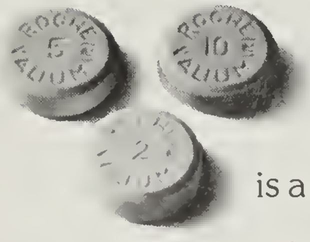
A character all its own.



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The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

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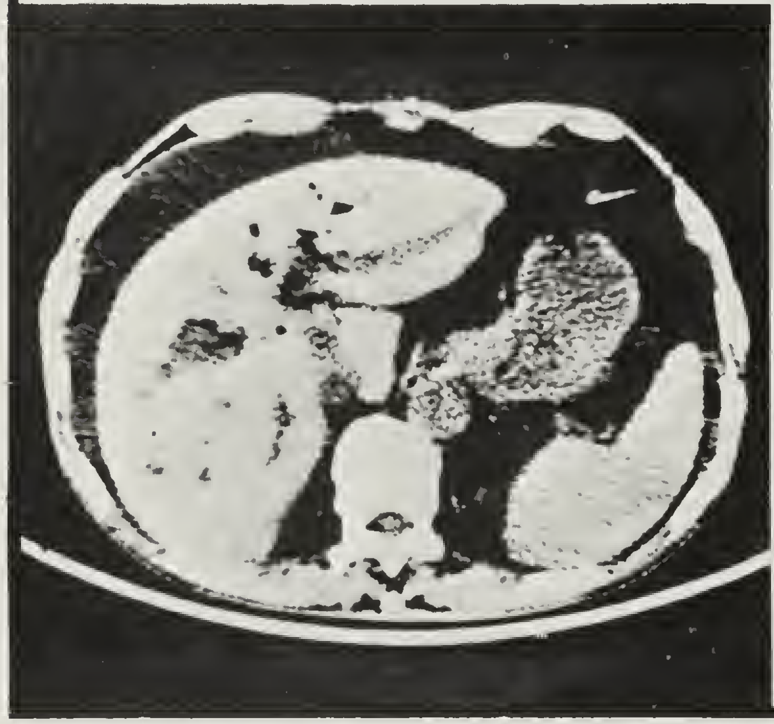

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in the treatment of

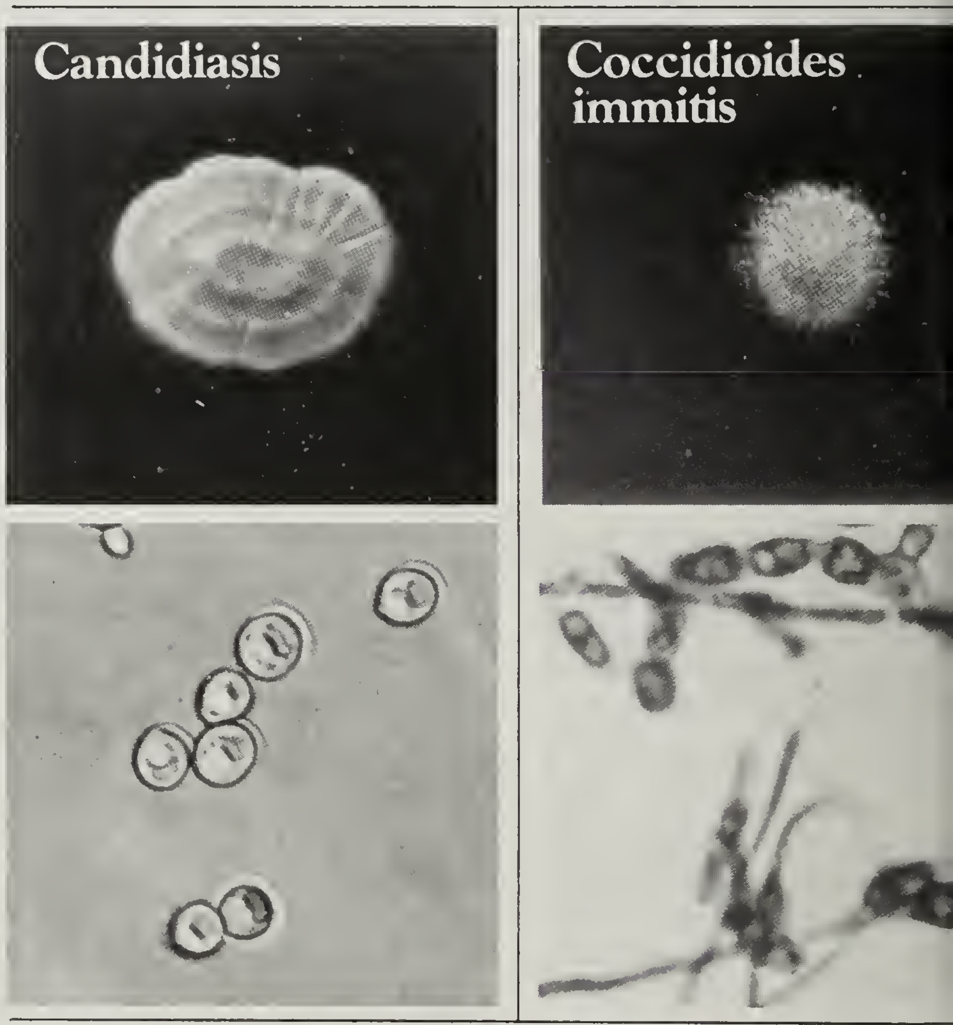
Efficacy of MONISTAT i.v. therapy...

highly favorable rates of recovery or clinical improvement

- effective against systemic candidiasis
  - chronic mucocutaneous candidiasis
  - coccidioidomycosis
  - cryptococcosis
  - paracoccidioidomycosis
- in patients who did not respond to amphotericin B therapy
- in immunologically-compromised patients with severe infections

rapid onset of clinical improvement
In most patients who responded, improvement was observed within 1-3 weeks of MONISTAT i.v. therapy; however, prolonged treatment courses may be necessary to prevent relapse.

no development of fungal resistance during therapy reported
In vitro resistance of clinical isolates has not been observed during clinical trials.



Photos courtesy of Dubos, J. R., Bacterial and Mycotic Infections of Man, J. B. Lippincott Company, 1968 and Burrows, W., Textbook of Microbiology, W. B. Saunders Company, 19th edition.

and the side effect profile

no nephrotoxicity or other permanent adverse effects in clinical studies

- even in prolonged therapy
- even in patients with renal disease

extensively evaluated
During clinical trials in over 400 patients, no permanent adverse reactions were reported.

Adverse reactions
MONISTAT i.v. should not be administered undiluted. Rapid injection of the undiluted solution may produce transient tachycardia or arrhythmia.

Adverse reactions include phlebitis, pruritus, rash, nausea, vomiting, febrile reactions, drowsiness, diarrhea, anorexia and flushes. Transient hematologic changes have occurred; therefore, the patient should be closely monitored.

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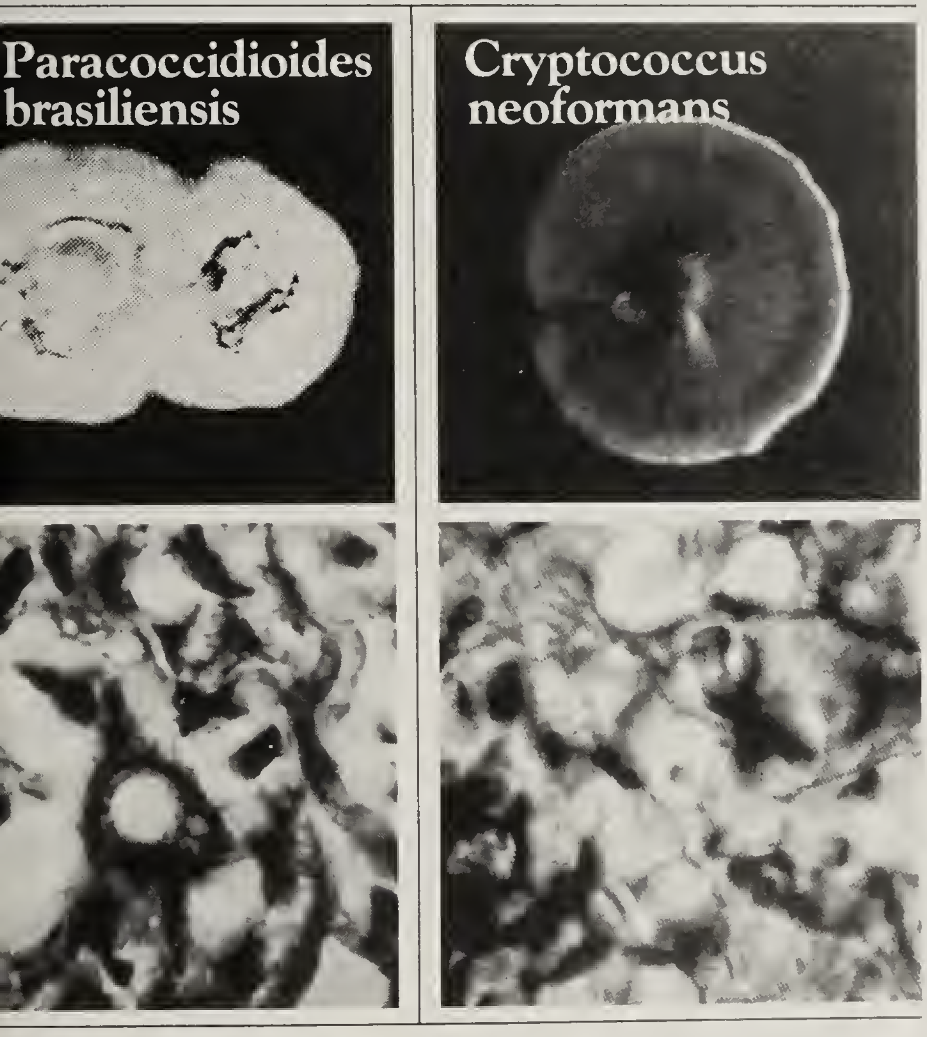
advancement systemic mycoses



generally well tolerated by severely debilitated patients

- cancer patients
- patients receiving immunosuppressive drugs
- patients with serious underlying conditions such as renal insufficiency or diabetes mellitus

for more information

Because of the uniqueness of MONISTAT i.v. only some of the highlights can be presented in this advertisement. For more information, i.e., product monograph, dosage schedules, etc., please fill out the coupon on the next page and mail to Ortho Pharmaceutical Corporation.

Please turn page for Prescribing Information

Ortho Pharmaceutical Corporation
Raritan, New Jersey 08869

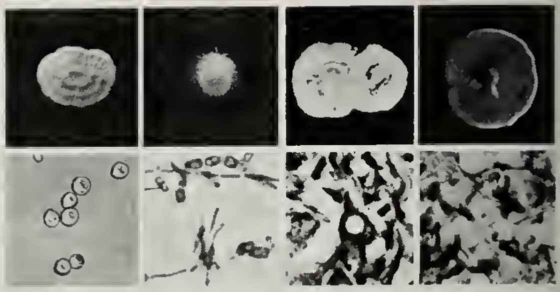
Monistat i.v.

TRADEMARK

(miconazole)

broad-spectrum activity against major systemic fungal pathogens

in systemic fungal infections...



Monistat i.v.

TRADEMARK

(miconazole)

a broad-spectrum systemic antifungal agent

DESCRIPTION: MONISTAT i.v. (miconazole) 1-{2-(2,4-dichlorophenyl)-2-[(2,4-dichlorophenyl) methoxyl ethyl} 1 H-imidazole is a synthetic antifungal supplied as a sterile solution for intravenous infusion. Each ml of this solution contains 10 mg of miconazole with 0.115 ml PEG 40 castor oil, 1.0 mg lactic acid USP, 0.5 mg methylparaben USP, 0.05 mg propylparaben USP in water for injection. Miconazole i.v. is a clear, colorless to slightly yellow solution having a pH of 3.7 to 5.7.

CLINICAL PHARMACOLOGY: MONISTAT i.v. is rapidly metabolized in the liver and about 14% to 22% of the administered dose is excreted in the urine, mainly as inactive metabolites. The pharmacokinetic profile fits a three-compartment open model with the following biologic half-life: 0.4, 2.1, and 24.1 hours for each phase respectively. The pharmacokinetic profile of MONISTAT i.v. is unaltered in patients with renal insufficiency, including those patients on hemodialysis. The in/vitro antifungal activity of MONISTAT i.v. is very broad. Clinical efficacy has been demonstrated in patients with the following species of fungi: *Coccidioides immitis, Candida albicans, Cryptococcus neoformans, and Paracoccidioides brasiliensis.*

Recommended doses of MONISTAT i.v. produce serum concentrations of drug which exceed the in-vitro MIC values for the fungal species noted above. Doses above 9 mg/kg of MONISTAT i.v. produce peak blood levels above 1 μg/ml in most cases. The drug penetrates into joints.

INDICATIONS: MONISTAT i.v. is indicated for the treatment of the following severe systemic fungal infections: coccidioidomycosis, candidiasis, cryptococcosis, paracoccidioidomycosis, and for the treatment of chronic mucocutaneous candidiasis. However, in the treatment of fungal meningitis and urinary bladder infections an intravenous infusion alone is inadequate. It must be supplemented with intrathecal administration and bladder irrigation. Appropriate diagnostic procedures should be followed and MICs should be determined.

MONISTAT i.v. should not be used to treat common trivial forms of fungal diseases.

CONTRAINDICATIONS: MONISTAT i.v. is contraindicated in those patients who have shown hypersensitivity to it.

WARNINGS: Rapid injection of undiluted MONISTAT i.v., may produce transient tachycardia or arrhythmia.

PRECAUTIONS: Before a treatment course of MONISTAT i.v. is started, the physician should make sure that the patient is not hypersensitive to the drug product. MONISTAT i.v. should be given by intravenous infusion. The treatment should be started under stringent conditions of hospitalization but subsequently may be given to suitable patients under ambulatory conditions with close clinical monitoring. It is recommended that an initial dose of 200 mg be given with the physician in attendance. It is also recommended that clinical laboratory monitoring including hemoglobin, hematocrit, electrolytes and lipids be performed.

It should be borne in mind that systemic fungal mycoses may be complications of chronic underlying conditions which in themselves may require appropriate measures.

Pregnancy: Reproductive studies with MONISTAT i.v. in rats and rabbits revealed no evidence of impaired fertility or harm to the fetus. There are no data, however, on the use of the drug in pregnant women.

Children: Since the safety of miconazole i.v. in children under one year of age has not been extensively studied, its benefits in this age group must be weighed against the possible risks involved.

ADVERSE REACTIONS: Adverse reactions which have been observed with MONISTAT i.v. therapy include phlebitis, pruritus, rash, nausea, vomiting, febrile reactions, drowsiness, diarrhea, anorexia and flushes. In the U.S. studies, 29% of 209 patients studied had phlebitis, 21% pruritus, 18% nausea, 10% fever and chills, 9% rash, and 7% emesis. Transient decreases in hematocrit and serum sodium values have been observed following infusion of MONISTAT i.v. Thrombocytopenia has also been reported. No serious renal or hepatic toxicity has been reported. If pruritus and skin rashes are severe, discontinuation of treatment may be necessary. Nausea and vomiting can be mitigated with antihistaminic or antiemetic drugs given prior to MONISTAT i.v. infusion, or by reducing the dose, slowing the rate of infusion, or avoiding administration at mealtime.

Aggregation of erythrocytes or rouleau formation on blood smears has been reported. Hyperlipemia has occurred in patients and is reported to be due to the vehicle. Cremophor EL (PEG 40 castor oil).

DRUG INTERACTIONS: Drugs containing cremophor-type vehicles are known to cause electrophoretic abnormalities of the lipoprotein. These effects are reversible upon discontinuation of treatment but are usually not an indication that treatment should be discontinued.

Interaction with the coumarin drugs resulting in an enhancement of the anticoagulant effect has also been reported. In cases of simultaneous treatment with MONISTAT i.v. and coumarin drugs, the anticoagulant effect should be carefully titrated since reductions of the anticoagulant doses may be indicated.

DOSAGE AND ADMINISTRATION

DOSAGE

Adults. The doses may vary with the diagnosis and with the infective agent, from 200 to 1200 mg per infusion. The following daily doses, which may be divided over 3 infusions, are recommended:

Organism	Dosage Range*	Duration of Successful Therapy (weeks)
Coccidioidomycosis:	1800 to 3600 mg per day	3 to > 20
Cryptococcosis:	1200 to 2400 mg per day	3 to > 12
Candidiasis:	600 to 1800 mg per day	1 to > 20
Paracoccidioidomycosis:	200 to 1200 mg per day	2 to > 16

*May be divided over 3 infusions.

Repeated courses may be necessitated by relapse or reinfection.

Children: A total daily dose of about 20 to 40 mg/kg is generally adequate. However, a dose of 15 mg/kg body weight per infusion should not be exceeded.

ADMINISTRATION: MONISTAT i.v. should be diluted in at least 200 ml of fluid. The diluent of choice is 0.9% sodium chloride or alternatively Dextrose 5% injectable solution. The intravenous infusion should be given over a period of 30 to 60 minutes.

Generally, treatment should be continued until all clinical and laboratory tests no longer indicate that active fungal infection is present. Inadequate periods of treatment may yield poor response and lead to early recurrence of clinical symptoms. The dosing intervals and sites and the duration of treatment vary from patient to patient and depend on the causative organism.

OTHER MODES OF ADMINISTRATION: Intrathecal. Administration of the undiluted injectable solution of MONISTAT i.v. by the various intrathecal routes (20 mg per dose) is indicated as an adjunct to intravenous treatment in fungal meningitis. Succeeding intrathecal injections may be alternated between lumbar, cervical, and cisternal punctures every 3 to 7 days. Bladder instillation of 200 mg of diluted solution is indicated in the treatment of mycoses of the urinary bladder.

HOW SUPPLIED: MONISTAT i.v. is supplied in 20 ml ampoules.

MONISTAT i.v. (miconazole) is an original product of Janssen Pharmaceutica N.V., Belgium, and co-developed by Ortho Pharmaceutical Corporation.

Ortho Pharmaceutical Corporation
Raritan, New Jersey 08869

Ortho Pharmaceutical Corporation AM-9-79
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Please send me more information on the broad-spectrum, systemic antifungal agent... MONISTAT i.v. (miconazole).

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October 1978

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Quinamm™

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Brief Summary

INDICATIONS: For the prevention and treatment of nocturnal recumbency leg muscle cramps, including those associated with arthritis, diabetes, varicose veins, thrombophlebitis, arteriosclerosis, and static foot deformities.

CONTRAINDICATIONS: Because of the quinine content, Quinamm is contraindicated in women of childbearing potential, in pregnancy, in patients with known quinine sensitivity, and in patients with glucose-6-phosphate dehydrogenase deficiency. Hemolysis (with the potential for hemolytic anemia) has been associated with a G-6-PD deficiency in patients taking quinine.

PRECAUTIONS: Thrombocytopenic purpura may follow the administration of quinine in highly sensitive patients. Recovery will follow withdrawal of the medication.
Cinchona alkaloids, including quinine, have the potential to depress the hepatic enzyme system that synthesizes the vitamin K-dependent factors. The resulting hypoprothrombinemic effect may enhance the action of warfarin and other oral anticoagulants.

ADVERSE REACTIONS: Aminophylline may produce intestinal cramps in some instances, and quinine may produce symptoms of cinchonism, such as tinnitus, dizziness, and gastrointestinal disturbance. If ringing in the ears, deafness, skin rash, or visual disturbances occur, the drug should be discontinued

DOSAGE AND ADMINISTRATION:
1 tablet upon retiring. When necessary, 1 additional tablet may be taken following the evening meal.

Product Information as of September, 1977

U.S. Patent 2,985,558

Merrell
MERRELL-NATIONAL LABORATORIES Inc.
Cayey, Puerto Rico 00633

Direct Medical Inquiries to:
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Division of Richardson-Merrell Inc.
Cincinnati, Ohio 45215, U.S.A.

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8 3305 (Y371A)

for Knotts in the night

Quinamm™

each tablet contains quinine sulfate 260 mg., aminophylline 195 mg.

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See opposite page for prescribing information.

Does it influence your choice of a peripheral/cerebral vasodilator*?

- Vasodilan—compatible with coexisting diseases
- Vasodilan—compatible with concomitant therapy
- Vasodilan—compatible with your total regimen for vascular insufficiency

*****Indications:** Based on a review of this drug by the National Academy of Sciences-National Research Council and/or other information, the FDA has classified the indications as follows:

Possibly Effective:

1. For the relief of symptoms associated with cerebral vascular insufficiency
2. In peripheral vascular disease of arteriosclerosis obliterans, thromboangiitis obliterans (Buerger's Disease) and Raynaud's disease.

Final classification of the less-than-effective indications requires further investigation.

Composition: Vasodilan tablets, isoxsuprine HCl, 10 mg. and 20 mg. Vasodilan injection, isoxsuprine HCl, 5 mg., per ml

Dosage and Administration: Oral 10 to 20 mg., three or four times daily Intramuscular. 5 to 10 mg. (1 or 2 ml.) two or three times daily Intramuscular administration may be used initially in severe or acute conditions.

Contraindications and Cautions: There are no known contraindications to oral use when administered in recommended doses. Should not be given immediately postpartum or in the presence of arterial bleeding.

Parenteral administration is not recommended in the presence of hypotension or tachycardia.

Intravenous administration should not be given because of increased likelihood of side effects.

Adverse Reactions: On rare occasions oral administration of the drug has been associated in time with the occurrence of hypotension, tachycardia, nausea, vomiting, dizziness, abdominal distress, and severe rash. If rash appears the drug should be discontinued.

Although available evidence suggests a temporal association of these reactions with isoxsuprine, a causal relationship can be neither confirmed nor refuted Administration of single dose of 10 mg intramuscularly may result in hypotension and tachycardia. These symptoms are more pronounced in higher doses For these reasons single intramuscular doses exceeding 10 mg. are not recommended. Repeated administration of 5 to 10 mg. intramuscularly at suitable intervals may be employed.

Supplied: Tablets, 10 mg., bottles of 100, 1000, 5000 and Unit Dose, Tablets, 20 mg., bottles of 100, 500, 1000, 5000 and Unit Dose; Injection, 10 mg. per 2 ml ampul, box of six 2 ml. ampuls.

U.S Pat. No. 3,056,836

VASODILAN®

(ISOXSUPRINE HCl)

20-mg tablets

MeadJohnson PHARMACEUTICAL DIVISION

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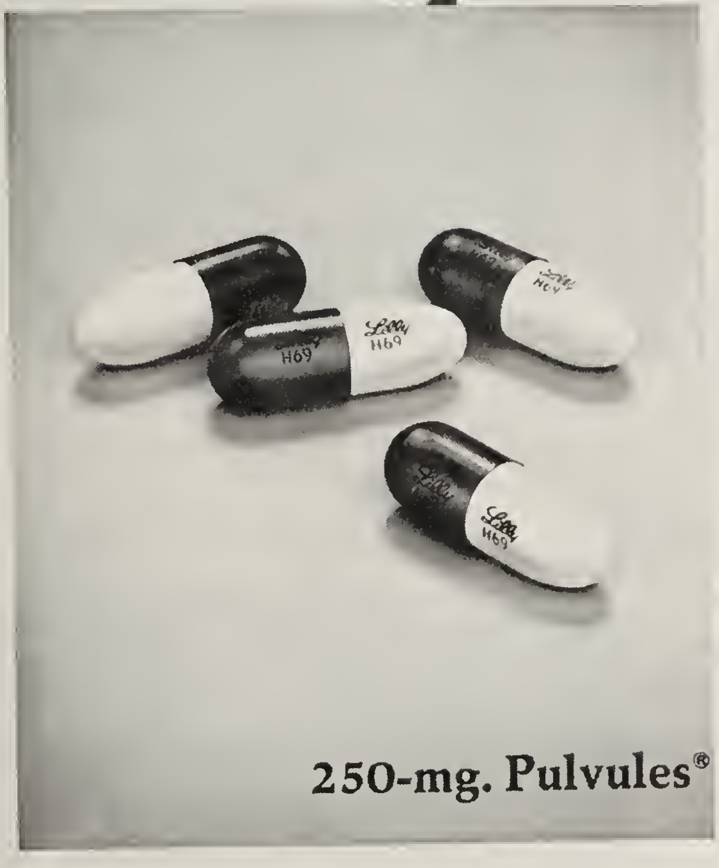

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Dr. R. Patterson Russell, Associate Professor of Medicine at Johns Hopkins Medical Institution, Director of the Hypertension Clinic at Johns Hopkins Hospital and Chairman of the High Blood Pressure Commission of Maryland, state the following in the March, 1979 issue of "Preventive Medicine," the official Journal of the American Health Foundation:

> **"Blood pressure measurements by the Vita-Stat correlated as well as did measurements by physicians and provided reliable determinations suitable for the purposes of screening, referral and monitoring of treatment."**

Introduction

William B. McGrath, M.D.

When I arrived in Phoenix in 1948 Bill McGrath was one of the four actively practicing psychiatrists in Phoenix. I was number five. Then, as now, he was not only well liked by all (both colleagues and patients) but respected as well.

He was and still remains a medically and neurophysiologically oriented psychiatrist, not only an excellent diagnostician but a versatile and perceptive therapist as well.

Though of eclectic orientation he was sufficiently skeptical to avoid the many "new" techniques, not to say fads, which perennially pop up like weeds in the over-fertilized garden of psychiatry. The methods he did adopt and apply were well selected and, as psychiatrists go, he was among the more effective.

His air of modest, unassuming affability, enhanced by his vivid analogies and vocabulary (modern dancing is "vertical epilepsy", "love is a verb") result in a rehetorical style which is as precise as it is poetical.

His (now 100) essays are not merely palatable but often spicy food for thought. I hope he'll have time to write a hundred more.

Richard E. H. Duisberg, M.D.

Erotomania - de Clérambault's Syndrome or Psychose Passionelle

Otto L. Bendheim, M.D.

Definition

The fixed delusional belief that a member of the opposite sex has fallen madly in love with the subject and has proposed, even consummated marriage. It is usually a respected, successful man of a higher socioeconomic class, who is the object of the erotic phantasies.

Scenario No. 1

The spacious, rather neglected and dreary grounds of a large western state hospital. Paper bags, cigarette butts, empty coke cans and other refuse strewn all over the dusty, weed rimmed pathways. Seated on the benches, lying on the brown sunburned grass or strolling dejectedly up and down: the most pitiful remnants of society, some severely retarded, some senile, some showing the motionless faces of the burned out, vegetating psychotics.

In ths midst of this inferno, a radiant exception, a very bulky, old black woman, waddling up and down, with a happy, amiable smile, engaging in joyous and animated conversation with anyone she can get hold of, never mind that no one pays any attention to her. No one ever replies.

She is quite a sight; to the front of her ancient many-patched dress of uncertain origin and style and supported by an ample bosom are pinned all matter of junk: old shoe laces, safety pins, labels of shoe boxes, a key chain, dozens of pieces of cheap jewelry in disrepair, ribbons, medallions, campaign buttons, five crucifixes dangling from a rusty chain, and other assorted souvenirs.

What does she say to her unresponsive audience? "A nice day, ain't it? I'm on my way home to my husband, Dr. S- - -, I'm cooking him a fine meal tonight. You all know he is my husband and you must call me Mrs. S- - -. See you all later. I'm late now."

With that she would direct her attention to the next little group, assuring them with pride and persistence of her dignified and prosperous role in marriage and society.

The facts: Dr. S. was a young staff physician at the hospital who had never exchanged more than a few cursory words with the patient. His wife was about his own age, some 40 years younger than the other "Mrs. S."

The ending. There is none. The patient died many years later of obesity, hypertension and old age, happily convinced that her husband, whose house she had never seen, was truly and eternally hers.

Scenario No.2

Sunday, the Day of the Lord. As the afternoon approaches we witness the miracle of the magic renewal, the ritual of transfiguration and blissful union - the marriage ceremony -uniting once again the world famous band leader, crooner and purveyor of musical schmaltz, appearing on the TV screen every week at the appointed hour, watched and adored by millions of hypnotized Americans, but giving himself in marriage to his one and only true wife. She had waited for the moment all week, lived for nothing else.

She had patiently performed the monotonous duties of a lowly file clerk in a governmental office, hired many years ago under one of the federal programs for the handicapped.

Handicapped she was. She stood about four feet, six inches; her prominent hunchback protruding almost to the height of her head. Her face distorted and asymmetrical, her legs curved and of unequal length, the tragic residual of infantile polio before the days of vaccination.

She had spent several hours this Sunday morning, like every other Sunday, in preparation of the solemn and happy occasion, her hair done up and over the towerlike contraption, which give her eight inches of additional height, hiding the top of the kyphotic curve from her lover on the screen. Her white lace bridal dress, adorned with pale pink lace roses, had been made years ago by the city's most prestigious and expensive dressmaker, at a price equal to three month's salary.

The shoes were so high heeled and fragile that our bride could hardly stand up, not even with the aid of her crutches, let alone walk. The penetrating scent of heavy perfume fills the room. On the coffee table are the champagne glasses and a bottle of French champagne in a wine cooler.

She is now ready for the marriage ceremony. She turns on the TV, the crooner appears. A happy, blissful expression transforms the bride's homely face — the union takes place aided by her rapid, masturbating movements of hands and thighs.

The ending? There is no real ending. After several years of weekly matrimonial bliss with her hero, who

---

From: Camelback Hospital, 5051 N. 34th Street, Phoenix, AZ 85018.

appeared on the screen only out of true love for his little "wife", she dies prematurely of heart disease.

Scenario No. 3

A pleasant, intelligent, attractive woman, comfortable, relaxed, not a bit anxious. She informs the psychiatrist at once that she really does not know why her family physician insisted on the consultation. There was absolutely nothing wrong with her. She had never felt better. Her story was simple. After 15 years of unhappy marriage to a man who (she felt) was below her intellectual and educational standards, she had suddenly found out that her family physician had fallen madly in love with her, was thinking only of her, and was going to divorce his wife, leave his family and marry her. How did she know all that? "I just know," with a superior smile. She readily admitted that there had been no improper conversation or contact between her and her beloved, not one word had ever been said, but it was obvious that they were meant for each other. It was just a matter of time.

After several totally unsuccessful sessions, after complete failure of various neuroleptics, the patient did not return until ten years later, when she was brought in by her children, presenting the identical clinical picture of obsessive and obstinate conviction of being cherished and loved by a different man she had seen only once or twice, who hardly knew her but who was constantly on her mind and, she was sure, she was on his mind. She had forgotton about her old family physician but her present lover was undoubtedly going to be her future husband. He was a successful, prominent business man, happily married, who was totally unaware of his "involvement." This patient remained happy, satisfied with her platonic state. (Her dreams and daydream phantasies were far from platonic.)

Nothing happened. They lived happily ever after, but not together.

Discussion

These three cases present erotic delusions of a strange nature. There is no suffering, no embarrassment, not the slightest hint of resentment about the world's failure to acknowledge the "facts" of the marriage or prospective marriage. Delusions of grandeur in paranoia are usually associated with a bitter struggle for recognition and, failing to be recognized as the exalted person the patient believes himself to be, he usually becomes aggressive, intolerant, litigious or pugilistic. He slanders and insults those who refuse to give him his due.

The manic-hypomanic with delusions of success and achievement invariably acts out his beliefs. He is not satisfied with a platonic affair but demands (and occasionally forces) consummation. He is under tremendous pressure, is hyperactive, restless, aggressive, impatient, unable to postpone his immediate sexual and other needs.

Erotomania lacks the cyclical and genetic features of the manic depressive illness. It lacks the ambivalent insecurity, the autistic isolation and affective blunting of schizophrenia.

It differs from adolescent hero worship or adoration of matinee idols in that the latter is always a conscious and elaborate effort of the enamored to gain attention in full realization that this attention by the love object, while ardently desired, is not yet achieved. The adolescent realizes that she has to compete with thousands of equally ardent girls, and that the idol pays very little or no attention to her.

De Clérambault, the French psychiatrist, who described erotomania, considers it a special category of paranoia. However, contrary to the negative, unpleasant and distressing aspects of paranoid, persecutory or referential delusions, erotomania is conceived by the afflicted as no hardship whatever, rather as a great blessing, a wonderful, often ecstatic experience leading to fulfillment of the most intimate wishes and desires, quite directly so, not indirectly or second-hand, as in delusions of reference.

De Clérambault's syndrome differs from the grandiose-delusional identity change of the paranoid in that the patient does not assume a different identity. She does not become one of the great lovers or heroines of history. She does not become St. Mary or Joan of Arc. She remains herself, only changes into a new marital or amorous status.

However, she shares with other paranoids the rigidity, systematization, invulnerability toward reality and confrontation, the superior attitude and inflexibility seen in these conditions.

The dynamics of this situation are not too difficult to analyze., The patient, always a female, has been short changed in life by socioeconomic or racial repression, by crippling disease, by an unhappy marriage or generally speaking by lack of wish fulfillment. In order to preserve ego integrity, she substitutes a phantasy lover. Immediately the delusion becomes so fixed and reality-resistive that the patient is transformed into a state of heavenly bliss.

Treatment has been futile. Psychotherapy, neuroleptic medication, electric convulsive therapy, behavior modification, etc., have been to no avail. The patient has a capacity to defend herself successfully against the sobering, saddening effects of reality, and continues in her happy phantasy world to such an extent that it would appear almost cruel to interrupt her dreams.

The occasional embarrassment to the imagined "lover" is another matter. Usually the absurdity of the claim is so obvious that no particular defense is necessary. There however are exceptions.

In summary, de Clérambault's syndrome, or erotomania, is a rare systematized delusion of an erotic nature, which has some of the earmarks of paranoia and of manic grandiosity but lacking some of the more malignant features of these conditions. It is treatment resistive.

Observations on Healing and the Physician's Role

Thomas E. Bittker, M.D.

Illness disturbes the whole person including his interactions with his environment. Increasingly, research confirms that conflict, under or over stimulation, inability to cope with negative feelings, and loneliness correlate with susceptibility to disease, delayed healing and increased mortality. Consider: 1) Men with "Type A" behavior are much more likely to develop coronary artery disease over a ten-year period than those not showing this pattern.[1] 2) Stressful life experiences have been positively correlated with onset and severity of chronic illness, the frequency of exacerbation of illness, and with delayed recovery.[2] 3) Loneliness contributes to heart disease, perhaps mediated by the increased vigilence and increased corticosteroid and catecholamine discharge experienced by the individual unprotected by the bond of family, friends or tribe.[3] 4) Chimpanzees torn from their band, feed poorly, lose weight, and relinquish their will to live. 5) Infants separated from their mothers and offered care in sterile, but emotionally cold hospital environments will soon develop morasmus. 6) Demographically-controlled studies indicate that compared to their married cohorts, the widowed have a four-fold increase in mortality in the year following the death of their spouse.[3]

Conversely, positive attachments mitigate against the effects of stress and disease. Critical to successful recovery from major surgery, for example, is confidence in the ability of the surgeon. Indeed, in a sample of 100 open-heart surgery cases, this confidence had a higher correlation with successful outcome than did the American Heart Association classification of severity of heart disease.[4]

Hope facilitates healing. Persuasive evidence for this principle is offered by a study performed in a rehabilitation program in a veteran's administration hospital for patients with chronic neurological disease.[5] The rehabilitation program was designed primarily to catalyze the hope of patients by changing the hospital's emotional atmosphere from that of a warehouse, where patients were expected to remain until dead, to that of a rehabilitation center directed toward restoring patients to useful lives. Hospital staff scrupulously attended to each patient by carefully evaluating their status; frequently adjusting treatment regimens; and attempting to re-establish the bond between the patients, their families, and prospective employers. On first evaluation, all of the eighty patient-participants warranted the label of "chronic". All had been hospitalized a least three years; some had been bedridden and were incontinent. Within three months after the initiation of this program, 70 percent were able to leave the hospital; after two and one half years, 30

From: Arizona Health Plan, P.O. Box 5000, Phoenix, AZ 85010. Dr. Bittker is Director of the Division of Health Maintenance Services.

percent were self supporting.

Efforts to reduce preoperative apprehension can markedly shorten morbidity. In a controlled study of children about to undergo tonsillectomy, 22 children received only routine postoperative care, whereas an experimental group of 22 were, in the company of their mothers, compassionately oriented to the procedure.[6] A nurse-educator explained the operation and helped each child anticipate how he or she might feel after it. In the process she attempted to allay the aprehension of both parent and child. At the end of one week, half of the experimental group had been successfully discharged with minimal postoperative morbidity; all the children who did not receive this interview remained hospitalized.

As Dr. Jerome Frank points out, the most powerful healing emotion is "expectant faith".[7] The physician is in an ideal position to mobilize expectant faith. His power to heal emanates from two sources. First, he occupies a social role of respect, power and trustworthiness similar to that of a parent.

A patient in distress is likely to view his physician with the attitude of trust and dependency that a small child feels toward a good parent. Second, his treatment is based on a coherent theory that expresses the world view of the society in which both he and the patient function. Since this shared world view makes sense out of life, it reinforces the sense of group belonging and thus combats the demoralizing sense of isolation that typically accompanies illness.

The physician's power to heal is related to the placebo effect—operating in from 30 to 60 percent of cases—to accentuate the healing impact of a medical or surgical intervention. The placebo effect is directly correlated with the degree of patient-physician rapport and the intelligence of the patient. Its impact occurs regardless of the power of the medication or of the operation with which it is being compared. Indeed, some argue that the improved lifestyle enjoyed by coronary bypass patients, in the face of no significant improvement in long-term survival, may be related largely to the placebo effect.

Healing is a product of the therapeutic partnership between the patient and the physician; this perspective on healing is productive whether or not one is confronting acute or chronic distress. Unfortunately, our culture reinforces the view of physicians as providers of short-term intervention. Often, physicians are seduced by patient demands to provide relief of long-term problems using short-term, incisive, "fixes". Thus, we may perform unnecessary surgery or prescribe addicting medication, in an effort to provide relief for problems which are the consequence of a lifetime burdened by bad habits, anxiety, anger and low self-esteem. Although the power of our short-term repair techniques may be limited when confronted with unhealthy lifestyles (smoking cessation will have a greater impact on lung cancer rates than will successful lung surgery); our capacity to facilitate healthy behavior is nonetheless immense. Physicians retain a position of enormous prestige in society. In spite of recent political assaults against organized medicine, ones personal physician typically is listed at or near the top of opinion polls assessing status. Over 40 percent of patients claim that they would exercise more or smoke less if only their physicians would make these requests of them. What follows is written to serve as a guide for physicians wishing to facilitate patient change.

Be Willing to Listen

In order to better understand our patient's life position, it is important to hear him out. When listening, try to comprehend how the patient is living his life outside of your office. Listen also for what the patient is requesting of you. Reflect back to the patient what you thought you heard him say so that you both have a shared understanding of his problem and his perceptions.

Define the Limits of Responsibility for the Problem and for its Relief

Your responsibility as a physician is to offer your knowledge and skill in facilitating healing while the patient is under your immediate care. Recognize that no matter how skillful you are in the operating room or how broad your knowledge of pharmacology, in most cases your efforts will be predicated on the patient's willingness and capacity to follow your instructions outside the office. Patients must be enlightened as to their power in accelerating or undermining recovery. Specific options would be made clear. Many of our problems reflected in "informed consent" controversies revolve around our inability to clarify responsibility issues with patients.

In further defining responsibilities, consider: 1) What is it that the patient wishes to achieve or change as a result of his interaction with you, the physician? 2) What is the patient willing to risk or relinquish in behalf of his goal? 3) How and when are you and the patient going to assess the progress of treatment? 4) Having considered all of the above, how much are you willing to invest in behalf of the patient's request?

To illustrate, let's consider the example of the patient who suffers hypertension. Ideally the patient should have as a goal: 1) the reduction of long-term risk for morbidity and mortality secondary to hypertension. 2) He should be willing to invest the effort and time required to take medication regularly; the loss of spontaneity in his diet associated with adhearing to a low-salt regimen; and money necessary to pay for medicines and for your fees. 3) An ideal follow-up assessment should occur during your next appointment with the patient when you take his blood pressure, assess side effects from medication, and you review together his home-blood-pressure log. 4) Having considered the patient's willingness to participate in this treatment regimen, you might then indicate your willingness to care for the patient. In some instances treatment contracting fails, because the patient's expectations are not supported by his willingness to work in behalf of these expectations. In these instances, the burden of responsiblity for success falls on you. Ultimately, when treatment fails, both you and the patient will be disappointed. When contracts are poorly understood and when the consequences of failure are significant in long-term disability or death, liability suits commonly occur.

Stay Involved

Take the initiative in establishing follow-up appointments for patients rather than waiting for their symptoms to provoke the need for such appointments. Aggressive follow-up programs convey to patients that they will not be abandoned. Furthermore, patients are more likely to comply when they know that you are both interested and available at a specific time for them to report .

Involve the Family when Appropriate

Many of our efforts will require the patient to make lifestyle changes. The family can serve as an important ally in obtaining maximum cooperation.

Review Specific Lifestyle Options with the Patient

Common, culturally encouraged indescretions in lifestyle include: 1) A reliance on high-salt, refined carbohydrate, saturated-fat, and caffeine-laden diet. 2) Lack of exercise. 3) Tobacco dependency. 4) Alcohol excess. 5) Absence of regularly-scheduled relaxation or pleasurable activity.

Positive modification of the above lifestyle factors will improve longevity in middle-aged adults by almost 10 years.[8] Although it does not require a physician's skill to recognize lifestyle problems, it may require a physician's prestige and interest in order to encourage the patient to make lifestyle changes.

Use Outside Professional Supports in Behalf of your Healing Objectives

Visiting nurses, physical therapists, clergymen, teachers and social workers can all serve as excellent guides to the patient's recuperation. Solicit their involvement and you will ultimately be rewarded by increasing patient cooperation. Working with these professionals will require a careful review of your instructions and will represent some sacrifice in time. On the other hand, for many problems such as pediatric allergy, the rehabilitation of the pain patient; or recovery from major surgical procedures; progress will be critically affected by these outside professional supports.

Develop a Healing Belief System

Be sensitive to all aspects of your communication—verbal and non-verbal—knowing that your messages shape patient expectations. Furthermore, be aware that patients may be so anxious during office interviews that they may not be able to hear you accurately. Office anxiety may be reduced and adherence to instructions may increase, if you supplement your office consultations with letters or written material intended to amplify the details of your treament program.

Consider Homework Assignments

Provide homework to patients if such homework is something that the patient can readily master and has relevance to treatment. The hypertensive patient, for example, may be requested to monitor his blood pressure at various times during the day to determine diurnal fluctuations and to assess impact of drug treatment. Similarly you may request specifically that the patient remove the salt shaker from his table and substitute some other form of seasoning. Parents of an allergic child may be instructed to change furnace filters once a month and offered an environmental check list for antigens. Before assigning homework, determine what the patient is willing to offer in the way of cooperation and effort. Provide a written checklist or request a daily log regarding patient success at homework activities. Review this at the time of the next visit.

Individualize Your Expectations

An active, cooperative patient will have a much brighter prognosis than a resistant, demanding, overly-dependent patient. Nevertheless, taking into consideration the limits of the latter patient, you can enjoy success, as long as you avoid the pitfall of assuming too much responsibility for the patient's care. Alert the patient and his family as to the outcome-options available to them predicated on the level of cooperation and effort that they put into the problem. Reinforce the patient for whatever positive behavior he is able to demonstrate in behalf of a positive outcome. Avoid anger when a patient fails to cooperate. If you find yourself becoming angry, it is quite likely that you are assuming too much responsibility.

In spite of the immense technological advances witnessed in medicine during the past generation, the artful medical practitioner can never forget that he represents only one half of the partnership in the healing process. We can succeed only when the patient agrees to participate as our partner in treatment.

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Nuclear Hysteria

Hal J. Breen, M.D.

Before attempting to explain 'Nuclear Hysteria' it will be necessary (to borrow a legal term) for a proper foundation to be laid. Otherwise, one is open to the charge that what purports to be an explanation, however convincing, refers to nothing. In short, what is meant by 'Nuclear Hysteria' and what evidence is there that there is such a thing?

Hysteria, in the sense used here, does not refer to any of the formal types of reference or usage of psychiatric nosology, but to the older and non-specific usage which is totally descriptive, and known to everyone. Nevertheless, there are some etiological characteristics of the state to be described which are shared with hysterical types of character disorders and with conversion hysteria. What is meant by 'Nuclear Hysteria' is a mental state characterized by greatly hightened affects, of such magnitude that the usual modulating effects of various ego functions such as reality testing, the sense of reality, the regulation of drives and impulses, congnitive processes and judgement are impaired. For example, the impaired reality testing may lead to statements that are generally characterized as "exaggeration" because they ignore the ordinary constraints imposed by reality. The impaired sense of reality may give rise to statements implying some degree of dissociation, such as "I have lost all sense of time. . . It is like a nightmare", etc., (a reported reaction to the Three Mile Island nuclear accident). Impaired drive control is manifested in impulsive flight or destructive behavior which may be directed toward external objects or people, or turned inward, even to the extent of suicide. Ordinary cognitive processes, such as the distinction between an atomic explosion, and other types of explosions, of vastly less destructive nature, were commonly ignored in reaction to the Three Mile Island accident.

What, then, are the affects which can bring this state about? The most obvious correlary is with the panic state, in which ego functions are overwhelmed by fear, so that ordinary adaptive functions are preempted by the need to flee from danger, or in the most severe cases bring about a total catatonic paralysis.

What needs to be explained is the nature of the fear; the reasons for the inordinate hightening of the fear, but most importantly the relationship of these two aspects to the affect of pleasure, a feeling which would not

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From: Professional Psychiatric Services LTD., 3352 East Camelback Road, Ste B, Phoenix, AZ 85018.

ordinarily be associated with a panic state, but is surely associated with 'hysteria' and serves to differentiate the latter from ordinary panic. It would strike any ordinary observer as remarkable that situations involving the most indisputable and severe hazard to life frequently do not inspire panic, and this is in contrast to 'Nuclear Hysteria' where the immediate hazard to life is minimal. Soldiers in combat do not ordinarily succumb to panic, at least collectively. It has been noted that there was a remarkable dearth of panic reactions during the London blitz, and that psychiatric hospitalization actually declined. In World War II, during a bombing raid over Hamburg Germany which lasted for an entire week, the children continued to attend school (in subways). The viability and capacity for adaptation of human beings under the most extreme stress has been demonstrated innumerable times, individually and collectively, and requires no proof, in the 20th century.

It is clear, then, that some factor other than the existance of an external danger is necessary in order to bring about an hysterical panic state. The dynamics of the conversion type of hysteria contribute to the further understanding of 'Nuclear Hysteria'. In this situation, the symptom, such as paralysis of a limb, is the symbolic representation of a compromise between conflicting wishes. For example, a patient developed a disabling paralysis which she described as feeling "dead", after an automobile accident in which she at first thought that she had killed another girl. The paralysis did not develop until she was forced to relinquish her conviction of homicide. The anamnesis revealed that the patient had taken over the maternal role in her family when her mother had died, at age thirteen; and that her father, after five years, had remarried a girl her own age, who had proceeded to assume the role of mother in the family. The paralysis, in this case, was the symbolic representation of being "dead", an unacceptable homicidal wish directed toward the usurping stepmother. The strength of the symptom - paralysis - can only be understood as a response to a powerful unconscious wish, therefore containing an element of gratification and pleasure.

And so it is that the fantasies associated with 'Nuclear Hysteria' are quantitatively and qualitatively distinguished from the ordinary panic state by the pleasurable element of wish fullfillment, which is implicit in them.

Case History

This is an articulate and intelligent twenty eight year old professional, who was interviewed because he was known to be actively engaged in the antinuclear protest movement. Nothing else was known about him, prior to the interview. He had logical and seemingly rational reasons for his position (he was opposed to all forms of nuclear development, both military and non-military), but behind this could be discerned two major types of fantasies. He was particulary concerned about the possibility of poisoning, from atomic waste, and he connected this primarily with fear of eating or drinking "hot waste". This anxiety was exacerbated by the fact that the radioactivity was not detectable by smell or taste, the most common and reliable guards against poisoning. The (coprophagic) implications of this were obvious. The oral aspect of his fear was further highlighted by his association of radioactive poisoning with cancer. He spoke of being "eaten up" with cancer. The second major type of fantasy concerned being burned. At various times he spoke of being "burned to a cinder", incinerated, fried to a crisp and sometimes this was associated with total destruction, such as being "vaporized."

In his professional life, he seemed to derive satisfaction from his conviction that he was dauntless in his opposition to the forces of evil. The latter was represented, however vaguely, by powerful "interests" and the adumbration of conspiracies and unseen connections. His personal life was "liberated" and somewhat shallow, in that he cherished his "freedom" and was quite explicit in his denial of a real committment to the girl with whom he had been living for about six months.

If one assumes that the fantasies expressed by those who are most disturbed by nuclear technology represent a symbolic bridge to the earliest oral and anal developmental anxieties to which we are heir, then the questions must arise as to why this is so, and how such fantasies can in any way represent the consummation of a wish. The nexus of the problem must lie in the ambivalence which is intrinsic to every constraint which is imposed on an instinctual drive or wish. Consider, for example, the impulses which very young infants must have toward destruction by devouring, or cannabalism. The infant, after all, can see that what he eats disappears, and it must seem to him that it is destroyed. We see residuals of this, in projected form, in the playful assertion of the adult (toward the young baby) "I could just eat you up." It is undeniable that the fear of being eaten is ubiquitous in the human, and in our everyday speech we speak of things being "gobbled up" as a synonym for being destroyed. We may assume, then, that what fuels the fear of being eaten is the unconsummated cannabalistic wish. And so it is with the constraints imposed on the excremental functions.

The wish to avoid the terror of losing a part of one's self can lead to withholding, in infancy, with the association of fantasies of bursting and exploding. The fear of loss of excremental control recalls sensations of burning, as in the eneuretic's dreams of fire.

Fecal matter, through the imposition and development of constraints on the where and how of elimination, comes to be perceived as bad, dirty, and above all else as poisonous. In this way, elimination is seen as a way of getting rid of everything inside that is "bad" and conversely, retained feces is seen ultimately as a source of poisoning. In the early 1900's it was quite seriously believed that schizophrenia was caused by constipation. A popular detergent, for example, is today advertised as killing "toilet bowl germs." Originally, however, the feces is seen as something valuable and a part of one's self. Young children not uncommonly express real anxiety over flushing of the toilet and the disappearance of feces, during the toilet training period. In general, there is always some resistance to the modificaiton of drives, because every modification of a drive involves a constraint or repudiation of a wish. The implication is that the wish is never totally destroyed; and behind the fantasies of being eaten by cancer or being burned, destroyed or poisoned which are at the core of nuclear hysteria are the repudiated but nascent oral and anal wishes of infancy.

Having explained this much, the question arises as to whether anything significant has been added to our understanding. After all, everyone has essentially the same legacy of repudiated infantile wishes, but it is obvious that not everyone is driven by a fear of cancer, poisoning, or burning. The essential question remains: Why the prevalence, quite worldwide in scope, of nuclear hysteria at this particular time?

The answer, I believe, is to be found in those social circumstance which have altered the development of the superego in very profund but little understood ways, over a period of at least several generations. The superego has its ontological origins in the very earliest instinctual renunciations. The character of the superego is determined by diverse and complicated forces, but may be said to hinge on two central processes. First is the necessity for externally imposed constraints on sexual and agressive drives. Secondly, there is the process of assimilating or introjecting the constraint as one's own. Social changes which interfere with either of these processes would be expected to modify the character of superego, with reverberating consequences for intrapsychic stability and interpersonal relationships. The child, for example, is taught by the mother (or mother surrogate) to defer gratification of the wish for instant fullfillment of excretory impluses. The loss of feces, originally a source of anxiety, becomes acceptable through the development of an attitude toward them which exaggerates negative feelings (a reaction-formation). It is not denied, of course, that there are inborn maturational sequences which contribute to the acquisition of internal control of drives, but this does not diminish the essential role of experience in this achievement. The movement from an externally imposed constraint to an internal constraint occurs within the framework of a loving and gratifying dyadic relationship, and events which seriously disturb, or preclude, such a relationship results in impaired superego development. Given the achievement of internalized constraints, what is the fate of the drive which is constrained? We say that it is "repressed" or otherwise modified, but there has not been very much concern with the social consquences of that portion or aspect of the drive which endures. Freud wrote about the "repetition compulsion" and the "return of the repressed" but his most seminal treatment of the subject was in *Civilization and its Discontents*[1] in which he elaborated that thesis that "each instinct renunciation" implied a modicum of guilt, and he felt that the burden of guilt had ominous implications for the future of civilization. He did not treat in any formal or systematic way the mechanisms which determine the "socialization" of derivatives of repressed drives, particularly as this might be influenced by modes of interaction between mother and child, or by cultural change. It is in these areas that we see the disturbances which give rise to 'Nuclear Hysteria'.

Eric Ericson in a speech delivered at the University of Delhi in 1963 stated that "Mutual understanding between infant and mother is the primal source of all moral motives" and that "All moral, ideological and ethical propensities depend on the early experience of mutuality"[2]. The establishement of what he calls "basic trust and confidence" is thus the precondition for the establishment of the superego. Ericson's conceptualizations coincide with the observations of many others, such as Greenacre,[3] Sandler,[4] Mahler,[5] Bowlby[6] and many others. There is less agreement, and less is known about the specific ways in which the development of basic trust and confidence might be impaired, from a developmental standpoint. Even less is known about later influences which might impair a developmentally established diathesis for a benign and trusting relationship to others.

What is indisputable is a world-wide drift toward fewer internalized constraints. This has taken the form of repudiation of what was formerly proscribed, but more than this, has been expressed as a metaphysical rebellion, in the denial of any authority for restraints or proscriptions.[7] The elaboration of reasons for this movement is beyond the scope of the present paper, but two aspects seem particularly perinent to the symptomotology of nuclear hysteria. The most obvious and perhaps the most profound change in terms of development vis a vis the mother (and the father) is in the advent of 'science' as a source of authority in child rearing practices. The central superego precursors of constraints and approval in the parents, so far as these rely on 'science' are preceived by the parents and indirectly by the child as coming from without, as something "external" to the parents. It is important to realize that this is a very recent phenomenon, coincident with the decline of religion and tradition as sanctions for child rearing practices. The "knowledge" of child rearing practices which is based on religious or traditional sanction is experienced as something intuitive and internal, and so an integral part of the parental identity which forms the core of the superego "I". Not only are the proscribing aspects of superego function affected in this way; the forgiving and approving functions, essential for the development and maintainance of self esteem, are affected in a similar manner. What the parent does not perceive as an inner conviction, but as something tentative, arbitrary and coming from without is thus not included in the introjected parental imago which becomes the core of the superego. Consequently, there is no internalized conviction of being "bad", or at least very "bad". By the same token, there is no firm conviction of the possibility of approval, forgiveness or atonement. The intrapsychic mechanisms for ensuring a harmonious balance betwen freedom, responsibility, guilt and forgiveness thus become blurred and confusing. The parallel phenomenon on the social level is in the decline of social influences which determine or reinforce proscribed behavior, and the consequently diminished importance of tradition or social attitudes toward guilt, atonement and forgiveness. The central role of religion as a socially sanctioned means of ameliorating guilt was recognized by Freud, himself an atheist. Freud considered that the amelioration of guilt was the most important "purpose" served by religion. It would be an error however, to assume that these forces alone - changes in parental attitudes toward what is proscribed and what may be forgiven, and the radically evolving social attitudes toward the authority of tradition and religious beliefs - have by themselves determined the evident repudiation of internalized constraints. From the standpoint of later experiences, as they influence superego functioning, it is postulated that even well established basic trust and confidence is vulnerable to those experiences which cannot be reconciled with such an attitude. To the extent that people identify with each other and with groups, we are all influenced by the behavior of others, so that in the aftermath of the bloodiest century in human history, it is more difficult to maintain a benign and hopeful attitude toward others. Furthermore, the existance of the superego itself may be jeopordized in those situations where the level of guilt is intolerable to life. This was well known during World War II, when Nazi officers, unable to function in the way demanded of them (on the Eastern Front), were transferred to duty in the death camps. After some months, they could be transferred back to combat, with the expectation that their conscience would no longer be a hinderance in the carrying out of orders.[8] In these situations, it would appear that the only possibility for survival was to "destroy" superego functioning. If the superego is the heir to inhibited aggression,[1] then untrammelled aggression would be the consequence of superego destruction (the repudiation of internalized constraints).

The diminution in internalized constraints diminishes guilt, but at the same time makes the achievement of self-approbation more difficult. Every step in the acquisition of control over instincts must be balanced by some intrapsychic means for dealing with the hostility consequent to the frustration of the "repressed" wish. Thus, the child can only learn bowel control as a means of consolidating a positive affective relationship to the mother. The distortion of intrapsychic mechanisms which integrate and meliorate the consequences of frustration, together with the decline of social structures which serve the dual purposes of prohibiting and approving determines the behavior which we have labeled "Nuclear Hysteria". It is not an accident that what the nuclear hysteric perceives as 'bad' in nuclear development - the possibility of poisoning and contamination - is a projection of those forces within us which generate unexpiable guilt, the cannabalistic and anal-sadistic impulses which first gave rise to our superego.

The same distortions of intrapsychic mechanisms determine the lack of basic trust and confidence and the hightened suspicion which characterizes the nuclear hysteric; it is only through forgiveness that we are able to overcome distrust and guilt, and our mechanisms for achieving forgiveness are impaired.

Fantasies of atomic cataclysm and doom, like the world destruction fantasies of schizophrenic disintegration, represents a projection of the nuclear hysteric's own destructive impulses, untempered by the faith and trust which demands an unimpaired superego.

The realistically cataclysmic possibilities of nuclear war coincide with these fantasies, and the loss of faith in the possibility of redemptory "goodness" is like the anisotropic loss of innocence engendered by our 20th century history of holocaust and horror.

"World's end's not bangs or whispers
Dissolving pristine ice in polar seas
An unobserved attrition but spent our hope
Prostrate in holocaustal fires
Our gracile trust consumed in shattering blood and dust
Not Dresden's speckled thrush on wing to bring
Poor gutted faith viaticum."

The suggestion then, is that superego development is ontologically defective because of vague and poorly defined parental constraints which are not an integral 'part' of the parent. The basic trust and confidence which is a prerequistie to superego functioning, aside from all other influences, has been battered by collective experiences which have exacerbated guilt to an intolerable level. Finally, the social devices for ameliorating guilt (such as atonement through religious ritual), or the maintainance of hope through redemptory processes, has been largely lost. In this situation, threatened by the return of his own projected guilt - engendering hostility, the nuclear hysteric opposes all exploitation of nuclear energy, to ward off guilt and to preserve what remains of his sense of worth.

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Guilt and Shame

James E. Campbell, M.D.

"Guilt upon the conscience, like rust upon iron, both defiles and consumes it, gnawing and creeping into it, as that does which at last eats out the very heart and substance of the metal."

South

"I never wonder to see men wicked, but I often wonder to seem them not ashamed."

Swift

Some authors discuss shame and guilt as one entity while others see these as very different aspects of experience. Shakespeare, a prolific writer who has commented on guilt, said "The mind of guilt is full of scorpions." "Better it were that all the miseries which nature owns were ours at once than guilt." "They whose guilt within their bosom lies, imagine every eye beholds their blame." "Though it sleep long, the venom of great guilt, when death, or danger, or detection comes, will bite the spirit fiercely." "Suspicion always haunts the guilty mind: the thief doeth fear each bush and officer." Guilt is considered a rather formidable pursuader when it comes to directing human behavior." According to Horace Bushnell, "Guilt is the very nerve of sorrow." Blair said "Adversity, how blunt are all the arrows of thy quiver in comparison with those of guilt." Shame also carries a rather extensive regard by some; Platius concluded "I regard that man as lost, who has lost his sense of shame." Maria Edgewood adds "Shame is nature's hasty conscience." Thompson declared, "Of all evils to the generous shame is the most deadly pain."

The individuals who comes for therapy often carry their own visions of shame and guilt. For most, guilt is the feeling that they have when they believe they have done something wrong, are possibly going to be punished, or will evoke someone's displeasure. Shame, a less frequently used description of oneself, is the term conveying exposure of our faults to others. Traditionally shame and guilt have important psychological differentiations that should be kept in mind by the clinician if he is going to understand patients' communications. Patients, being unaware that "shame and guilt" describe different personal experiences, do not use these terms clearly.

Most authors agree that shame and guilt emerges somewhere between the ages of two and five.[1] Eric Erickson, in his *Eight Ages of Man*, sees shame as rising out of the second stage which he calls "Autonomy versus Shame and Doubt". Autonomy being synonymous with control and a wish to have a choice. Shame arises from immaturity in dealing with life situations, or lack of ability to develop complete control. Erickson states, "Shame is an emotion insufficiently studied because in our civilization it is so early and easily absorbed by guilt. Shame supposes that one is completely exposed and conscious to being looked at: In one word, self-consciousness. One is visible and not ready to be visible; which is why we dream of shame as a situation in which we are stared at in a condition of incomplete dress, in night attire, 'with one's pants down'." Shame gives way in the third stage of development called Initiative versus Guilt. "Visual shame produces auditory guilt which is a sense of badness to be had all by oneself when nobody watches and when everything is quiet - except the voice of the superego."[2] Initiative is approaching what seems desirable and hoping for new responsibility. It includes things such as planning and doing for the sake of being active. The conquest becomes pleasurable in itself. The association with guilt emerges because initiative often brings one into conflict with others in such a way that one's capabilities are over-stepped. It is exemplified in the competitiveness of the small child with the adult. With initiative comes fantasies of one's powers and confrontation with one's vulnerability, a combination often leading to the castration complex and an intensified fear of failure. Guilt may be associated with many different factors and most agree that it has elements of disapproval, fear of punishment and loss of love. The anticipation and dread of forthcoming consequences makes it a painful experience. Guilt may be the product of conscious or unconscious conflict. When unconscious it tends to stimulate abnormal behavior whereas the more conscious the conflict the more one experiences the sense of pain. Guilt and shame are thought by some to have a socially valuable side - being a necessary component in an individual's achievement of a true sense of socialization. (ie, Swift).

In clinical practice it is best to view guilt and shame as different entities. Shame can be considered the body feeling that comes when an individual is unable to live up to their image or expectations of themselves. These expectations may be part of their family code, upbringing, values interjected from early family experiences or ideas developed over their lifetime. When

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an individual experiences a need to be perfect it results in a sense of shame about day to day accomplishments. Individuals unable to raise their behavior to the level wanted will experience a sense of disharmony called shame. Helping individuals over this sense of shame means raising their level of functioning to better match the image they have, or requires lowering the image so it more closely matches his capabilities. Where an individual believes he should be functioning is relative and many who function at an extremely high level still experience disharmony because their perfectionism leaves them feeling that they are not accomplishing what they should.

Guilt, can be seen as a body feeling that comes when an individual is unwilling to accept the cost or consequence of his decisions. Guilt in a practical sense has nothing whatsoever to do with the rightness, wrongness, goodness or badness of any given act. It is purely the end product of the way an individual preceives his action. All decisions should be composed of at least three parts: One is an evaluation of the alternatives, second is a consideration of the costs/rewards that a given action will bring, and thirdly an action. There is no decision when individuals unwilling to evaluate the costs experience a sense of guilt arising from day to day "decisions". For example, an individual who goes to Las Vegas and places money on the spin of a roulette wheel, will feel guilty only if he cannot afford the loss. An individual recognizing and accepting the possibility of loss will not be devastated like a person unwilling or unable psychologically to accept the cost. Costs are not all measured in money; a parent's disapproval, a wife's harrassment, a boss' displeasure all represent potential costs in making decisions. An individual not considering costs is setting up to develop guilt. Eliminating the experience of guilt requires learning to carefully evaluate what the potential costs are in any given decision to see if the psychological attitude to accept the costs is present.

Patients may be advised that if they cannot afford the costs of a given decision then perhaps they should not make that decision. (ie, If one cannot afford to lose one should not bet).

Summary: The mechanisms behind shame and guilt are quite different. Helping a person deal with these two different experiences requires few moments of time on a physician's part to elicit whether or not the individual is discussing shame or guilt. Even though the two may be co-mingled and may exist at the same time in a given individual, one must help the patient divide their experience into parts so they can apply the proper principal to bringing about a solution.

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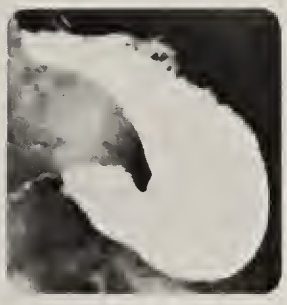

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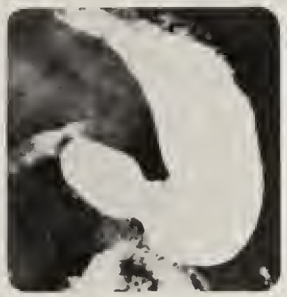

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†See Warnings, Precautions and Adverse Reactions.

See following page for prescribing information.

Reference:
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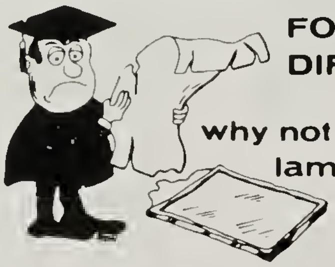

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Diagnosing the Troubled Child

Kent E. Durfee, M.D.

Although psychiatrists deal daily with emotionally disturbed individuals, most of us have at times felt a sense of frustration as we have searched for a diagnostic "label" that seems to "fit" a particular patient. There is even futher confusion and frustration if the patient's difficulty includes some degree of mental deficiency and one attempts to correlate the nomenclature of the American Psychiatric Association with that of the American Association for Mental Deficiency. If the evaluating person is not specifically trained in psychiatry, and if the patient happens to be a child, the difficulties mount in geometrical proportions.

The manifestations and symptoms of mental illness in children may differ from those of adults. For example, a depressed, unhappy child frequently acts out in an aggressive and destructive manner instead of presenting the more classic picture of vegetative signs, tearfulness and complaints of worthlessness so common at a later age. The resulting confusion frequently leaves the evaluating person with little opportunity to conceptualize the child's difficulties or to communicate meaningfully with others in his field.

It is hoped that a discussion of children, their needs and their psychological development, will be helpful to the general physician in recognizing and understanding the child in trouble and when a referral to a child psychiatrist should be made. Some of the differences between the mentally retarded, the child with minimal brain dysfunction, and the emotionally disturbed child will be covered, along with the kinds of things that can go wrong in the course of the child's growth and development that lead to his functioning below his potential. The subject of what makes the child what he is is extremely complex and multi-dimensional.

While we know that this world we live in is not a black and white one, and that there are very few absolutes, we all tend to think in dichotomies in an attempt to make some sense and order out of the various confusing shades of gray. For example, we talk and think in terms of good and bad, right and wrong, love and hate, mind and body, etc. Life is not this simple. If it were, the answers would be simple. An oyster might be traumatized by a grain of sand and form a pearl; is this good or bad? Value judgements depend upon whether the situation is seen from the point of view of the oyster or the person. In a similar sense, what is "good" for one person may be quite "bad" for another. Life involves trauma and stress. If the stresses are coped with adequately, the ego develops and matures; if they are overwhelming, the ego collapses. What is viewed by one person as an interesting challenge may be seen by another as an insurmountable obstacle that can only be dealt with through regression and illness. In the growing and developing child his abilities tend to be flexible and what may be seen as insurmountable one day may be seen as interesting and possible the next.

In thinking of the developing human organism, we frequently get caught up in two very important dichotomies: whether things are genetic or environmental and whether things are organic or emotional. At any given point in time a person is the sum total of the physical and mental characteristics handed down genetically by his parents, tempered and modified by the multitude of environmental forces which are impinged upon him. Thus, we are all both genetic *and* environmental beings. Physical illness affects us psychologically and our psychological state can and does affect us physcially. We are psychosomatic and somatopsychic beings. No one is totally healthy, nor is any living being totally ill.

Child development can be approached from the point of view of time, i.e., the critical periods of one's existence. Timing is important. There are specific times when traumatic events will have very important consequences in an individual's later development or

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lack of development.

At the moment of conception, all of the genetic factors are fixed. From this time on, whatever happens to the organism is due to environmental influences. We are learning more about the genetic causes of mental retardation. There may well be a genetic predispostion to minimal brain dysfunction. Some investigators also believe that genetic factors play a role in what is considered functional or emotional illness.

Since timing is important, frequently the earlier an environmental trauma occurs to the developing organism, the more important it is. The most important time of the pregnancy is the first trimester, and within this first three months, the most vital are the first five to eight weeks when the essential organs are being formed—heart, kidneys, and of course, the brain. Any severe maternal illnes during this time may lead to miscarriage and death of the fetus. Many things that may not result in fetal death, however, can result in severe fetal damage. We are all aware of the consequences of rubella during the first three months of pregnancy, x-rays to the mother can cause the child to microcephalic, and the drugs, such a Thalidomide, within the first three months can result in gross malformations.

Other things less dramatic may occur to the mother and to the developing embryo which in turn lead to less dramatic, but nevertheless important, permanent damage in the child. For example, the mother's addiction to alcohol or drugs, poor nutrition with avitaminosis, hormonal imbalances, and even the common cold during the first trimester, may cause an ectodermal dysplasia with congenital brain malformation and consequent mental retardation in the child. Many cases of mental retardation and minimal brain dysfunction are the result of this first trimester in utero damage. Minimal brain dysfunction is a syndrome involving a visual and/or auditory perceptual disturbance, frequently associated with problems of muscle coordination, hyperactivity, short attention span, and specific learning disability in a child of at least average intelligence.

If the genetic makeup of the fetus is adequate and the first three months of pregnancy are uneventful, the chances are then quite good that the pregnancy will proceed to term. The next critical period is the birth itself. A difficult labor with prolonged periods of fetal anoxia, abnormal positioning of the baby, inadequate maternal pelvic size, the use of instruments, etc., can cause birth trauma and central nervous system damage. Any later insult to the central nervous system such as tumors, infections, physical traumas, toxic conditions, etc., may result in conditions ranging from minimal brain dysfunction to brain damage with a loss of intellectual potential.

The environmental traumas to the otherwise healthy child need not be those which cause brain damage per se, but may be emotional and result in psychological fixation at a particular stage of development. The end result may be a severely emotionally disturbed child who functions at a retarded level without apparent organic cause.

In the first year of life one begins to separate himself from his environment. If the early mother-child relationship is adequate, the child can feel safe enough to tolerate the mother's temporary absence and gradually come to recognize that the mother is outside and separate from the self. Usually at three to four months of age, the child begins to recognize the mother or the mothering figure as a distinct and separate individual. If the mother-child relationship is for any reason inadequate, the child may not be able to tolerate the separation, and never be able to separate himself from her or from the other externals in his environment. Such a person may never develop a sense of himself as a person. Obviously, a child whose sense of self is so nebulous and fluid cannot develop adequately for the learning of later lessons. The early loss of mother or the mothering figure can result in a deep depression, wasting away, and death.

Between one and two years of age, if all has gone well, the child is beginning to learn certain aspects of control, not only of himself and his body, but also of the world around him. It is an age when the child learns the meaning of the world "no". It is a wonderful age—an age of magic when the child makes everything disappear by closing his eyes (and at times his ears). He can appear by stepping in front of a mirror and disappear by stepping away. If he is lonely, he can conjure up imaginary playmates. He can even control the heavens. On a moonlit night, he can walk and make the moon move; stop and the moon will stop. He learns not only what control he has over things, but what limits and controls the environment places on him. There is the gradual development of the primitive super ego or conscience, and through the conscience, the development of internal controls of his behavioral patterns. The child without internal controls may give vent to his impulses in an omnipotent and indiscriminate manner, with little or no concern for the rights of others. Until and as internal controls are established, the child needs external controls, limits and structure. Without control, he may feel quite helpless and become frightened and overwhelmed by his own anger.

At three and a half to five years of age, if the child has learned his earlier lessons adequately, he knows that he is a person separate and distinct from others, and that he is able to control certain aspects of himself and of his environment. He is then ready to begin to accept what sex he is. Children at this age beomce extremely interested in the difference between the male and the female, and the struggle with what it means to be a boy, or a girl. Providing the parents are secure in their own sexual roles and identites, the child can make a solid and adequate sexual identification with the parent of the same sex. If, however, the parents are insecure and unsure of their own roles, or if the sexual roles are reversed or distorted, the child may become confused in his sexual identity. This can lead to later unhealthy patterns with markedly disturbed interpersonal relationships. Depending on the nature and the severity of the child's difficulties and his methods of defending or trying to deal with them, he may be unable to pass comfortably on to the next psychological lesson, and he may continue through life hampered, frightened, and ineffective.

From ages six to twelve, the major psychological lesson to be learned is socialization or how one gets along with others. This is an age of competition with the self and with others, as well as learning to cooperate and work together in groups. There is a great deal of emphasis on the body and its development. It is important to see who is king of the mountain, who can jump the highest, spit the farthest, or hold his breath the longest. It is a time of best friends and strong loyalties. Teamwork and team competition become important and, of course, this is a time of tremendous emphasis on the learning of new facts and skills. During this time the child tends to give up thinking in specific and concrete terms, developing the capacity to abstract and generalize through the use of symbols. The healthy child is interested in school and the new knowledge he can obtain. The troubled child may be quiet, shy and withdrawn into fantasy, or angry, destructive and disruptive of others instead of being able to comfortably socialize with his peers and invest in his personal and academic achievement.

Adolescence is a biological and psychological phenomenon. The two usually go hand in hand, but not necessarily so. We think of adolescence as beginning at puberty and extending through the teenage years. With some, however, the psychological aspects of adolescence may occur prior to puberty; with some, it may extend far beyond the teenage period; and with others, it may never occur at all.

Biologically, the adolescent is undergoing tremendous physical and hormonal changes. Strange, new and frightening sexual urges and impulses occur. The adolescent must learn to live and to cope with these new impulses and, at the same time, do so while his physical appearance and bodily functions are rapidly changing. Thus, the adolescent is a stranger to himself and finds himself in the midst of an identity crises.

In the process of trying to get acquainted with his new self, the adolescent begins to questions many things. Who is he, what does he believe in, what does he want to become, what does he value, and does this agree with what he has been taught? There is a need to think things out and to decide for himself. The adolescent finds himself in turmoil and revolt where old values and ways of thinking and being must go by the board for the sake of the new. He must develop for himself an identity that is simultaneously compatible with his own historical past, with the ever changing present, and with his hoped-for future. If his parents are themselves mature and secure in their own way of thinking, feeling and believing, they can help their child through this difficult period of time. If, however, they are insecure, they may find themselves threatened by their child's rebellion and new ideas and stand in the way of his developing for himself a positive value system and an individual self-identity.

There are many subtle physical signs that frequently present in both the retarded child and the child with minimal brain dysfunction. They include such things as epicanthal folds, high-arched hard palate, malformed teeth, skull asymmetry, webbing between the fingers and toes, fetal palmar creases, short incurving little fingers, and the asymmetrical placement of the external auditory canals.

There is reason to believe that minimal brain dysfunction with perceptual difficulties is present in a fairly high percentage of the general population. A significantly large number of individuals with perceptual problems are also emotionally distured. They tend to express the underlying anger and frustration in a variety of ways, ranging from acting out in socially unacceptable behavioral patterns to schizoid withdrawal, fear and depression. It is quite possible that a large proportion of our socially unproductive citizenry are persons who suffer from minimal brain dysfunction. If so, early recognition coupled with remedial assistance might prevent the later development of dependent, defeatist attitudes.

There are, of course, many children, retarded, with minimal brain dysfunction, and emotionally disturbed, who manifest no outward physical signs. Diagnosing the troubled child is basically understanding where he is developmentally, from a physical, neurological, social, intellectual and psychological point of view. A child who is sufficiently atypical in any area of development warrants an evaluation in order to determine the nature and cause of his difficulties.

This evaluation is best accomplished through the team approach. The basic psychiatric team includes the psychiatrist, the psychologist, and the psychiatric social worker. As a physician, the psychiatrist assumes primary responsibility not only for screening the patient physically and psychiatrically, but also coordinating and integrating the findings from the psychologist, who utilizes a variety of testing techniques, and the psychiatric social worker, who works primarily with the parents in obtaining historical and developmental data. Additional diagnostic personnel frequently include the child's pediatrician or family physician, and the neurologist. The child's teacher, coach, scout leader, minister, etc., can frequently add valuable diagnostic information.

There are ways of determining whether a child's difficulties are predominantly emotional or predominantly organic. The history points the way for diagnostic confirmation through examination. An adequate history includes pertinent information regarding the grandparents, the parents and their siblings, as well as the siblings of the child in question. It should include not only medical data but information concerning the socio-economic status of the family, their religious, educational and ethnic backgrounds; along with the parental plans, ambitions, hopes and fears for the child in question. For example, the history might reveal that other family members had had difficulties similar to those of the child, pointing to one of the genetically caused metabolic or neurologic diseases. Information about other family members who have suffered or are currently suffering from a severe emotional illness should alert the physician to the fact that emotional factors may be contributing to the child's difficulties. Neurotic attitudes are frequently passed from one generation to another and often result in emotional illness noted in many members of the family. What are the current social or financial stresses within the immediate family setting? Children frequently react severely to environmental stress within the home without themselves being ill.

History of the pregnancy and the delivery is important. Early spotting or bleeding may be an indication of some early fetal damage. Maternal illness or trauma can result in impairment of the developing embryo, while a prolonged labor of difficult delivery may point toward organic brain damage.

The history of the child's early development is essential. Feeding problems are frequently noted in children who later show evidence of emotional disturbance. If the child learned to sit, stand, walk and talk at or near the appropriate age for these accomplishments, and later "regresses" or slows down without apparent organic cause, one suspects an emotional illness. On the other hand, congenital organic impairment is frequently manifested by a generalized delay in development.

Difficulty in learning to tie one's shoes or to ride a two-wheel bicycle may seem like inconsequential questions, but are signs of coordination difficulties and are often indications of visual motor coordination difficulties common in children with minimal brain dysfunction.

Soft neurological signs, hyperkinesis, muscular hypotonia, poor coordination, crossed laterality, immaturity, and a reversal in reading or writing, while not conclusively diagnostic, may strongly suggest minimal brain dysfunction.

While psychologists can and do utilize a wider variety of tests, much information can be attained through such tests as the Bender-Gestalt and certain portions of the Wechsler Intelligence Scale for children, which can help diagnose minor organic impairment. Emotional disturbance may be reflected in the inter-test scatter noted on the intelligence tests. Emotional problems are reflected in bizarre sexual or aggressive projections on the Rorschach test and the Thematic Apperception Tests. A child's drawing can be diagnostic. They may be pleasant, cheerful cephalopods (organic), or bizarre monsters with fire or electricity coming from their heads (emotional).

In the psychiatric examination, among other things, one looks for age appropriate play and verbalizations. If the child functions inappropriately, does he do so in a simple, "young" manner, or in a chaotic, disorganzied and bizarre manner? A short attention span in a hyperactive child is frequently an indication of organic impairment, while over-aggressive play, a hyperconcern over death, extreme fearfulness, or frank destructiveness of toys and play equipment are more frequently associated with emotional disturbance. Speech impediments offer diagnostic clues. Speech involving sound omissions are more apt to be organic, while sound substitutions are much more frequently associated with emotional disturbances. How does a child relate to the examiner? Does he reach out for a relationship in a friendly, albeit simple, manner, or does he tend to stay emotionally withdrawn and uninvolved? The latter is more frequently noted in the emotionally disturbed child. The examination of the child should be done only after he has been prepared for it. These preparations need not be lengthy nor involved. A matter of fact statement by the parents evidencing their conern over his "unhappiness" and a statement to the effect that he will be seeing a doctor (or a group of doctors) who will try to help them so that they in turn can help him, will usually suffice. The child knows that he is unhappy and is often surprised and reassured to find that the parents also know and want to do something about it. Any special procedure should be explained to the child in simple terms and in advance. The examination should include whatever laboratory work is indicated, a physical examination along with a basic neurological examination, psychological testing and the psychiatric evaluation.

While these are general guidelines used in determining the primary cause of the child's difficulty, there are going to be many children who remain confusing. Even with complete workups, we may not, in individual cases, be able to state for certain whether a given child's difficulties are predominantly organic or emotional: Frequently emotional problems compound and confuse an underlying organic condition.

THere is far more that we do not know about what makes a child what he is than what we do know. Further research in medicine, psychology and education, along with further work in the related areas of the social and biological sciences, may give us the answers we now lack. The diagnostic team is of vital importance because it brings together people with a similar interest but with different training, different experiences and different points of view. Just as the child's troubles are complex and multi-dimensional, a multiple disciplinary approach offers the best hope for an eventual solution; however, the earlier a pathological condition is recognized, the more expedient and effective is the treatment.

Flooding, Flashing & Flushing:

Emotional Repercussions

Sarah B. Nelson, M.D.

Introduction

"Flooding, Flashing & Flushing" is an admittedly flashy phrase designed to get the attention of the reader. Women have often used these complaints to get the attention of their doctors. This paper might have been more properly entitled "Some Aspects of the Emotional Problems Seen in Women Around the Time of Menopause". The real significance of the "Flooding, Flashing & Flushing" title is that the symptoms do not exist alone but are accompanied by emotional repercussions of at least equal importance. Flooding, or menorrhagia, may be related to a life-threatening cause and so produce fear or depression, but it may also be a nuisance or an embarrassment. Hot flashes and flushes, atrophic vaginitis, and dysuria are probably simply related to estrogen deficiency but can impact on many aspects of a woman's life with varying psychological consequences. Osteoporosis can be a disability of major proportions both physically and emotionally. In the past few years doctors have also become acutely aware of the iatrogenic emotional upheaval produced in women by the confusion and controversy over post menopausal estrogen therapy. The very women who were hooked on estrogen replacement therapy with promises of "feminine forever" are now often refused any estrogen therapy even in small doses for bonafide symptoms because of the apparently quite valid recent research findings about endometerial cancer. Doctors are often just as confused and unhappy as their patients. Nevertheless, they have become targets for women's anger, not only over the subject of estrogen replacement therapy, but also because of the broader issues of changing social roles and the evolving self-awareness of women today. The woman in the office is concerned about her personhood as well as her patienthood.

Defining the Menopausal Woman

First, just what is a menopausal woman and what is likely to be the emotional impact of her biological state?

From: 3352 E. Camelback Road, Phoenix, AZ 85018

She is above all, an individual, unique and special, not just an endlessly complaining patient plus or minus ovaries, plus or minus a uterus. She is a person who has about 25 to 30 more years ahead in which to work, to love, and to play. This is the end of her reproductive life, not the end of her productive life. This is a time for her to properly mourn her losses. This is also a time for reassessment, redirection, and renewal of purpose and meaning. To work constructively with this woman, stereotyping must be avoided. The popular image of the typical menopausal woman is negative. She is seen as exhausted, haggard, irritable, even bitchy, unsexy, impossible to live with, driving her husband to seek other women's company. She is considered to be irrationally depressed, unwillingly suffering a change that marks the end of her active life, an affliction to her friends and family. Actually she is a unique individual with her own personality patterns, her own intrapsychic equilibrium, her own heredity, her own past history of life experiences. She is currently dealing with her own special constellation of relationships and responsibilities. When she tries to envision her future it is filled with her own particular hopes, fears, and yet unknown realities. It is because of the infinite variety and complexity of these inextricably interwoven contributing factors that the signs, symptoms and sequelae of the physical menopause are so variable from woman to woman, and so often misrepresented and misunderstood.

Depression in Menopause

What are the common problem areas? The most common emotional symptom or syndrome is depression. This can vary from the inevitable emotional turmoil seen in all transition phases of life and responding to supportive measures, to severe incapacitating psychotic depression requiring hospitalization. This severe depression has until recently been termed an Involutional Melancholia. It is seen predominantly in women whose personality make-up is of the obsessive-compulsive type. Such a woman is neat and nice, conscientious and conforming. Some estimates are that one in ten women experience a severe depression during menopause, making it essential that doctors recognize and treat or refer these patients. It must be kept in mind that for women suicide rates are highest between the ages of 45 and 54. Women are in triple jeopardy for depression in this age group. There is first a suspected genetic vulnerability. Recent research suggests that certain more severe depressions seen in the involutional period (that is 45 to 55 for women, and 50 to 65 for men) may be transmitted as an X-linked dominant gene, explaining the 2 to 1 ratio of severely depressed women to men in these years. Secondly, the stereotypic social and psychological role of women has been one of learned helplessness and inhibition of anger, both contributing factors in the production of self-contempt and depression. By social concensus, anger in women has been inhibited, thwarted or diverted. Both men and women have been brought up with the idealization that "a lady" refrains from the expression of negative emotions. Anger has been permitted only in defense of helpless others. The prototype here is the lioness defending her cubs. Research findings demonstrate the presence of rampant sexual stereotyping or sexism in all of us, including psychiatrists of both sexes. Some identical behaviors have been demonstrated by research to be positively or negatively perceived depending upon whether performed by men or by women. Social norms support the woman who is more compliant, dependent, and passive in relationships with men. Aggressive, competitive, competent women may be seen as sick. They are often denigrated by the epithet, "castrating bitches". So it seems evident that the repression of constructive anger which could be useful in the service of a woman's rights and needs can be a social as well as a psychological cause of depression.

A third major etiology of depression is loss. Let us consider now a menopausal woman's multiple real, symbolic, and fantasied losses. First, there is the loss of the capacity to bear children. It needs to be re-stated that biological destiny does not define a woman, but her biological timetable does define her options. Although women today are rebelling against being perceived as "baby machines", the loss of reproductive ability probably has some impact on all women, as the unconscious lags behind social change. This loss will be differently perceived, depending upon whether it is a natural, surgical or premature menopause. For some it is equated with lost femininity, an end to youth and physical attractiveness; for others, an end to their womanly essence or reason for being; for the childless, the incontrovertible end to unfulfilled wishes. Other losses (shared with men in middle life) are the unfulfilled dreams of romance, fame and accomplishments, suddenly realized as probably no longer possible. Hopes of success and happiness fade, and as the psychological defense of fantasy life fails, depression may follow. Parents often become dependent or die in this time span, forcing the woman to face her own aging process and mortality. Children grow up and leave home, leaving an emotional void, frequently refered to as "the empty nest syndrome". Mother no longer has any clear place in her children's lives. Angered and saddened but unable to express her emotions, she may turn these feelings inward and become depressed. The depth of this loss depends upon the intensity of the investment of the woman in her children. If overly invested, their departure can cause shifts and stresses in the marital relationship also. The woman who previously derived much of her emotional gratification from her children, and also projected or transferred her marital dissatisfactions and conflicts on to them is now compelled to resolve her problems with her husband directly. He may be caught unprepared and be thrown off balance.

There are particular problems in regard to sex. A woman's opportunities for heterosexual sex may decline with divorce, death of her spouse, or the impotence of her partner. Sexual impotence is most commonly due to the three A's, Alcohol, Anxiety, and Anger. The woman's release from fear of pregnancy or from the nuisance of contraceptive measures may contribute to her increased interest in sexual activity just at the time when her man's interest is often on the decline. His reasons are multiple and varied. He may just be disinterested; he may be preoccupied with business; he may be depressed or drinking too much, or be diabetic. It is also possible that he may be having an extramarital affair for emotional support and to fulfill his own unexpressed dependency needs. All or any of these possibilities can add up to further feelings of loss and depression for the menopausal woman. Complications of such marital interaction abound. Even a retired husband at home can mean a loss in terms of personal space and routine to his wife.

One of the simplest and most common causes of intermittent male mid-life impotence is the normal physiological decrease of erectile capacity. This may become a source of growing resentment by a woman who misinterprets and magnifies this problem, rather than solving it herself in the obvious way by manual stimulation. This lack of understanding and communication can then lead to further loss for the woman. Depending upon her perspective, she will feel an absence of personal warmth or a loss of a sense of being needed or desired. Claude Steiner, a proponent of TA who wrote "Games Alcoholics Play", describes the menopausal woman as having a built up deficit in love, with depression coming from a lack of strokes, a lack of satisfying human relationships.

Other Repercussions

As has been outlined above, depression is a common emotional complication of the menopause. The depressions include Involutional Melancholia (more recently termed Psychotic Unipolar Depression), Manic-Depressive Illnesses, and Depressive Neurosis which is a much less severe and more mercurial condition. Additionally, other mental illnesses may be triggered or aggravated at this time. Those most likely to occur are alcoholism and other varieties of oral dependency, such as drug addiction and obesity. Schizophrenias may recur, as may paranoid states. This is also the time when a pre-senile organic psychosis, Alzheimer's or Pick's Disease may develop.

Then too, marital problems are myraid. A memopausal woman is ordinarily married to a 50ish male. Two people often grow, develop, and change at varying rates. It helps as marriages mature to have a shift in emphasis in the relationship away from performance and appearance, and toward companionship, empathy and sharing. Partners may instead grow apart, become weary, bored, feel they need a change. The sense of missed sexual opportunities may lead to a woman's breaking old social conventions by seeking out younger men or by finding pleasure for the first time in masturbation, or in unusual sexual fantasies, or in homosexual experiences. Then there is the "Gaugin Complex", in which the middle aged woman (or man) becomes a drop-out, giving up marriage, children, or career to go off to Tahiti, to a commune, or to join a religious cult.

Comments and Treatment Suggestions

One of the basic issues of menopause is to come to grips with the fact that no one can be fulfilled through someone else. A poignant statement from the jacket of a first novel by Ellen Schwamm titled *Adjacent Lives* state that each of us "must finally come to terms with the limitations of love in easing the imperfections and mysteries of the individual life". Interestingly, and perhaps surprisingly to some, mental health statistics show that marriage (rather than non-marriage and no children) especially the child rearing period is associated for women with less happiness, more stress, and more overt mental illness. In other words, married mothers may be unhappier than single, childless women! An idea borrowed from Anne Seiden, a psychiatrist involved in Research of the Psychology of Women, is that children should be looked upon as a time-limited occupational hazard with both positive and negative aspects. Perhaps some divorces at the time of the menopause can be seen as a disassociation by the woman from a failed attempt to derive her whole satisfaction from her relationships with husband and children. Additionally, it could be deduced that menopause and middle age ought to be a time of relief from stress with increased happiness and good mental health. And so it can be!

Dr. Ruth Moulton, Assistant Clinical Professor of Psychiatry at Columbia University has some points to make about the new feminism which pertain to all women including those who are menopausal. She asserts that it is not wise for doctors or anyone else to push a woman to work outside her home if she feels satisfied and productive there. But many women may use their children as a convenient protection against the anxieties of making a new or more independent life. Dr. Moulton lists four types of anxieties produced in women as they strive to make changes in their lives. These four syndromes are : 1) re-entry or entry anxiety 2) performance anxiety 3) self-assertion anxiety and 4) identity conflict anxiety. Traditionally in our society a woman's identity has been based upon her motherhood, femininity and sexuality. With attempts to change this identity, some of the feelings produced are feelings of guilt at not being available to the needs of others, fears of failure, fears of loss of approval and threats to security. Dr. Moulton suggests "as Therapists, we can only try to faciliate the change the patient envisions".

So with all this is it any wonder women become emotional when they are told by their doctors that their symptoms, whatever they may be, are simply due to "the menopause"? They are variously offended, insulted, angry, scared, tearful, anxious, distrustful. They need more than this kind of attitude. Women need our respect, our concerned interest, and our treatment based on the latest scientific knowledge. Most of all they need our perceptiveness to their individual needs. Listening to a patient doesn't mean letting her ramble on and on, making you impatient, and late for your next appointment. Active listening implies not only that you take time to listen, but that you guide the patient into areas of importance with appropriate questions, and that you try to be sensitive to what she is not saying, as well as to what she does say. Sometimes you may need to see someone else in her family to confirm her story or to clarify distortions. Sometimes your most important role is to help her reassess her potential and redirect her energies. She may be pretty much together but not know where to go from here.

Before concluding, it should be re-emphasized that the focus of this paper has been on "Some Aspects of the Emotional Problems Seen in Women Around the Time of the Menopause." The menopause has been blamed for a host of ills both physical and emotional. There has been no intention to place blame, but rather to attempt to clarify some relationships. Menopause after all does not occur in a vacuum, but rather in an individual woman usually in her middle years. Erik Erickson aptly termed the middle years "The State of Generativity versus Stagnation". Productivity and a sense of meaning can provide protection from the joylessness and weariness often seen in the middle years.

The image of middle life as the prime of life helps to offset its negative stereotype of decline. Good mental health at this time includes flexibility, ability to grow and change, as well as an acceptance of mortality. There is a need to depend upon skills and satisfactions rather than to try to outwit the normal aging process. Prevention includes 1) public education for all age groups and both sexes, 2) a positive attitude toward middle age with special attention to discussion of sexual functioning, 3) acceptance of satisfactions of daily living, and 4) an awareness of and attempt to modify personality make-ups in those who place undue importance on qualities of youth, or are unduly rigid, conforming, and conscientious.

Conclusion

It seems appropriate to conclude with a quote from Carl Jung, famous philosopher and psychologist. He said, "The serious problems of life are never fully solved. If it should for once appear that they are, this is the sign that something has been lost. The meaning and design of a problem seem to lie not in its solution but in our working at it incessantly. This alone preserves us from stultification and petrification."

To further illustrate Jung's point — here is a poem about creating a garden.

Life is full of rabbits in the night. When they see you have made a garden they come in and eat it up. Just remember, they never know what it is to make gardens."

So life may seem to deprive a woman of the products of her garden. She may experience the loss of uterus and ovaries, her children, her parents, her youth, her dreams, and even her husband. Nevertheless, like the gardener, she still has the capacity to work and to live creatively. As physicians, we have the opportunity and the privilege to help women appreciate and enjoy their potential for creative living at any age.

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Psychosocial Structure in China

F. Theodore Reid, Jr., M.D.

In this paper, I will share some observations and reflections about the People's Republic of China, based on a visit there in September 1978. The easiest way to do that is to contrast some of the issues and problems as they exist in the China with their counterparts in the United States.

Many of the comparisons and contrasts between American and Chinese cultures are not original. One of my friends is a Chinese-American anthropologist who has written extensively about American and Chinese cultures. I will draw heavily on his work. One important point, according to Frances Hsu is that Americans are very individual-oriented. The Chinese by comparison are very situation-centered, that is, responses and patterns are dependent upon the situations in which they find themselves, starting with the family.

It is important to start a discussion of psychosocial issues with the state of things prior to what the Chinese call "Liberation". Before 1949, more traditional Chinese culture existed and an examination of values must start there.

The Chinese family was a continuous stream in which one traced back to known and unknown ancestors and forward to children, grandchildren and unknown future generations. There was a sense of linkage, belonging and involvement in something that transcended the individual life. There was a belief that a good life that one was experiencing might be due to the virtues of an ancestor. Similarly, the troubles of this life might be helping an ancestor who was suffering in the Chinese equivalent of purgatory. In this framework, one's life and one's actions did not simply affect one's self.

This is in sharp constrast with the modern American family where there is discontinuity between generations. Rarely is there any significant involvement with grandparents and even more rarely with great grandparents. Such contacts when they do occur tend to be limited. One is reared by one's parents and in turn rears children, but at the point of college or the first job, there is a sharp decline in the involvement that a parent has with a child. This is in constrast to Chinese culture where grandparents are usually instrumental in the rearing of their grandchildren.

A second aspect of situation orientation relates to physical place. We may think nostalgically about where we were born and reared but Americans are mobile and therefore, geographically fickle. We don't feel bound to stay where we were born and we don't. In fact, we rarely visit, rarely keep contacts and we end up geographically rootless.

Another area of difference is the religious orientation in the two cultures. Religion in traditional China was like family structure, continuous, in which one's fate, in this life and afterlife, depended not only on one's own actions but on one's family's actions. Behavior had ramifications both for ancestors and future generations. Western religion presents us with a totally different situation. Our problem, religiously speaking, is to save our own souls. We are not responsible for saving the souls of our parents and after the age of either First Communion or Bar Mitzvah, we're not responsible for the souls of our children. That leaves us all isolated, not only in our families and our church, but also in the universe and with God. In the West, we stand alone.

These factors produce a kind of anxiety that is inherent in the West in general and especially in America, but is not a significant part of the Chinese experience. For instance, the Chinese adolescent has not been burdened either before or after Liberation with the choice of what to do with his life. Usually, it was clear that he would till the land since most Chinese have done that both before and since the revolution. However, if there were other alternatives, those choices were not the adolescent's. Parents, grandparents and now a commune committee make those decisions. This process is quite alien to American thinking but our way can be a burden. We have to choose the correct college and course of study at the age of 17 or 18. We feel we have to know what we want to do once and for all. The pressures on us by that age are great.

With intense involvement in any network, one's achievements and failures are distributed throughout that network. The highs get blunted and the lows flattened. Failure or success is shared in the network in which the Chinese have continued to live. By contrast,

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From: 7301 4th St., Ste. 4, Scottsdale, Arizona 85251

our successes and our failures are uniquely our own. It is not enough that one's father achieved professionally or financially because few Americans want to be known in those terms. One has to achieve in one's own right.

We have no network that says "there are other valued things for you to be involved with now". Enough is never enough for us because we stand alone and if we stand still, we do not know who will catch up and pass us. We are so achievement oriented that often we can't retire in peace.

There is data about how cultural differences affect us. Hawaii is one of the places where there have been studies breaking down mental illness by ethnic group. There, multiple cultures live in a similar setting so that the differences in mental illness presumably relate to family and cultural differences. In 1953, first admissions for Caucasians were twice that of Chinese in Hawaii. Chinese admissions for alcoholism were almost nil. The suicide rate was about double for Caucasians compared to Chinese. Statistically, admissions for organic psychosis were about equal, which indicates that the differences were probably not due to under reporting.

We shift now from Hawaii to the People's Republic of China. Initially, there was a denial that there was any psychiatric illness. "Liberation" cured everything—starvation, venereal disease, prostitution, drug abuse, parasitic disease, alcholism, etc. Recently there has been an acknowledgment of mental illness.

It is difficult for someone not able to converse to tell how much the old family structure has been undermined. One can only report impressions. It seems that the part of family linkage that had the religious element of ancestor worship, has been reduced. There is very little overt practice of religion of any kind in China today. However, in terms of the living family, one of the commonest sights in all parts of China is an elderly person with either an infant or a toddler. We visited a number of homes, all of which were multi-generational.

Impressions of psychiatry and psychiatric illness are based largely on a visit to a psychiatric hospital. It is a teaching hospital of about 100 beds, affiliated with one of the medical schools in Peking. The atmosphere was very similar to that in a comparable American teaching hospital.

In the absence of verbal language, our impressions were based on non-verbal interaction between staff and patients as well as transactions with the staff mediated through translators. We were unable to get statistics on mental illness in China. However, in that hospital, which they believed was typical, the majority of the patients (they guessed in the range of 80%) were admitted with the diagnosis of acute schizophrenia. (Indeed, they looked schizophrenic.) Staff estimated that about 20% of admissions were suicidally depressed. They do not hospitalize for neurosis. The incidence of organic illness was "quite low" and occurrence of senile illness was "rare". They attributed the latter to the involvement of the elderly in family life. Respecting and invoving the elderly in meaningful activities may indeed be a part of it. A second factor is that there is a great tolerance of the elderly. Some of the things that we would consider irritating and difficult to live with are comfortably tolerated in a culture less concerned about deviation.

In hospitals, schizophrenics are treated in multiple ways. They use "Western techniques", such as the major tranquilizers. In addition, however, they use purely Chinese medical techniques such as herbs and acupuncture, the latter predominately for agitation. They electrically stimulate the acupuncture needles, the treatment lasts about 20 minutes and terminates in sleep. The chemical treatment of last resort is insulin coma which they state is preferable to ECT, which they say "damages the patient." They use group therapy to help patients integrate into the unit and also to facilitate discharge. There is individual treatment, which we would call counseling. There does not appear to be an organized psychological theory base.

The follow-up network is quite enviable. The hospital is in the position to demand the presence of representatives of the neighborhood, farm or factory commune as well as members of the family. They are taught about mental illness in general and about how to treat the particular patient on his return to the community. Thus, the communes bear a degree of responsibility for reintegrating the patient into a setting in which he can function. The family, of course, has responsibilities for the discharged patient.

In addition to formal psychiatric care, there are some other related activities. Visiting a neighborhood commune, for instance, it becomes clear that "responsible people" who run the commune, feel free to intervene when they feel that a particular couple is having marital difficulties. What we think of as marital therapy occurs in the neighborhood in a mental hygiene framework. Another example was provided by one of our guides whom we got to know quite well. He had been a merchant seaman and later worked happily in a merchant seaman's hotel where he learned English. When English-speaking tourists came to China, there was a shortage of English-speaking tour guides. As a result, he found himself involuntarily transferred to Peking where he was unhappy. However, in "self-criticism meetings," he had gotten significant support from his fellow guides. They told him how important his job was, how much they liked him and pointed out the similarities between work with tourists and work with merchant seamen. In a month and a half, he had become much more comfortable with his job. A couple of years later, he was enjoying it.

He had had the equivalent of crisis intervention group therapy with his co-workers, which had avoided the need for him to enter the professional health care system. My impression is that these two examples are typical and that the demarcation that we make between professional and nonprofessional care gets blurred in China.

The state is involved in many aspects of life that we would consider highly personal. The state decides when people get married. Nobody is allowed to get married under the age of 25. The state is involved in family planning. After two children, couples are encouraged to use contraception, be sterilized or have an abortion. The state intervenes for the elderly who do not have family. They are housed and cared for in places called Houses of Respect and can be seen visiting various attractions with other sightseers. Of course, there are nursery schools and creches for nursing women in factories. There is group involvement in many things. One possible reason for the relatively low crime rate is that if a teenage were to steal a bicycle, all the neighbors would know he could not have bought it. Since everyone is aware of everyone else's life, there is less temptation to steal a bicycle. However, there is not a paranoid sense of Big Brother watching but is talked of as caring and mutual responsibility. (I remember when I was growing up and at an age when I did not have permission to cross the street. If I crossed the street, one of the neighbors was apt to call my mother.)

I have a limited number of reflections on China. I had a short visit, a limited knowledge of the culture and none of the language. In a general sense, their system seems to be working for them now. It provides a higher level of health care than in the past. They seem to be eating better. I did not see anyone in 6,000 miles of travel within China who looked malnourished and rarely saw anyone overweight. People smile on the street. They were curious and friendly. Those who could communicate did. There was no running away from foreigners, rather a sense of curiosity, interest and availability.

What I found myself doing on my return was reflecting about America with some concern about the price we pay for our brand of individualism. We appear to have mounting social disorder in our country. There is increasing stress disease, mental illness, family disorders and addictions of various kinds. We have a sense of unease and an absence of answers. We leave our parental homes literally or figuratively around the age of 18 to make our way. We get into early marriages, which tend not to work. We struggle very hard to get ahead except that we are frequently involved in motion without movement. We often confuse change with progress.

I came back with a lot of questions, particularly about my work as a psychiatrist. As a psychiatrist, one of my tasks is helping people adapt in a healthy way to their life situation, including their culture. I have some questions about our culture and the ways it stresses the individual. An individual can protect against illness due to stress with good friendship and/or family networks. However, our cultural values guarantee that there are few people who have them. Certainly, very few of the people I see in my practice have them. How can we build them within this culture? How do we achieve a sense of belonging, continuity and security in our lives without losing other things of value? I find myself reflecting more about America as I return from China.

Yogic Breathing and Psychologic States

H. J. Schulte, M.D.
V.V. Abhyanker, M.D.

Introduction

A familiar theme in medicine is re-awakening, certainly one that Sir William Osler would espouse. The core, concept involves a return to the wisdom of the ancients while concommittently exploring the expanding frontiers of sciences. One of man's oldest endeavors to control his physiology and well-being is the science and philosophy of yoga. Although yoga is not primarily a method of therapy much yogic literature, especially that of Hatha Yoga, indicates the curative and prophylactic value of many Yoga practices.

That changes in one's respiratory status produce physiological changes is a well documented fact. These physiological changes are accompanied by changes in mental status. Hyperventilation causes a decrease in blood Co_2, producing respiratory alkalosis. The familiar picture occurs of numbness and tingling in the exremities, dizziness, palpitations, muscle cramps, syncope and frequently a subjective sense of panic.[1] On the other hand the controlled reduction of respiration in yogic breathing produces a gradual level of accumulation of Co_2 which stimulates a cerebral circulation and aparently engenders mental tranquility and reduction of sympathetic activity in the Autonomic Nervous System. Students of yoga long ago discovered that control of respiration could be used to maintain health and to alleviate some illnesses.[2]

One of the authors (VVA) recently travelled to India and had the opportunity to interview several well-known yogic masters regarding their use of yogic breathing for selected emotional disorders. The goal of this paper is to discuss the psychologic changes associated with respiration controlled by the Yogic method.

Two case examples will be presented with discussion.

From: Psychiatric Residency Training Program, (Dr. Schulte, Director and Dr. Abhyanker, Senior Resident), Maricopa County General Hospital, 2601 E. Roosevelt, Phoenix, AZ 85008.

Yogic Breathing

Since 300 B.C., the philosophy of yoga has been based on the belief that the human body and mind are controlled by an immortal, super-human element in an called "Atma" or soul. Yoga is a Sanskrit word which means to mediate and to join the unenlightened (animal) nature of man to the super-human in him. This is accomplished through self restraint, study, various postures, control of vital breath, making the mind introspective, steadiness of mind, meditation and contemplation. These eight constituents of yoga can be practiced via the paths of action, devotion, mental control, and knowledge. Yoga aims for discipline of body and mind to maintain physical health, and to achieve higher spiritual awareness.

Yoga breathing (Pranayama) means voluntary regulation of breathing in a rhythmic fashion. This rhythmic breathing appears to induce a steadiness of mind and provides a controlling link between the body's basic neural activity and physiologic functions. Respiration is one of the few Autonomic Nervous System controlled functions that can also be easily controlled voluntarily. In order to more clearly grasp the concept of Pranayama it is useful to describe it.

The participant assumes a comfortable crossed leg position on the floor keeping spine, neck and head erect and letting the wrists rest prone on the knees. A circle is made by the thumb and index finger touching and pointing downward with the other three fingers straight. Pranayama is diaphragmatic breathing in which the abdomen is extended out during inhalation and contracted, pulling it inward, during exhalation. The subject should sit comfortably, breathing normally for a few minutes before proceeding to a comfortable, slow, deep exhalation. This is continued until the person has emptied his lungs of as much air as possible. There is a brief pause followed by slow, deep inhalation with the abdomen expanding as the diaphragm contracts and is pulled downward. The expansion of the abdomen with inhalation should be gradual and rhythmical and not abrupt or fast. Upon completion of inhalation there is a brief pause and exhalation is begun again. This cycle is repeated ten to fifteen times, usually increasing exhalation until it is twice as long as inhalation. Yogic breathing involves gradual reduction in the number of respriations per minute decreasing to twenty respirations in five minutes over a several week period.[3]

To aid the development of concentration during Pranayama, one school of thought recommends the use of visualization. The subject does the breathing exercise while facing a flower and thinking about the fragrance of the flower and the purity of air during breathing. For example, while exhaling one imagines that all bodily impurities are being expelled and that inhaling brings in fresh fragrant air energizing and nourishing the body. Practice in concentration is directed toward seeing the flower with the"mind's eye" during Pranayama.[4] An interesting aside is the growing interest in combining a relaxation technique and visual imagery as an adjunct to current medical treatments such as the work of Simonton's[5] with cancer patients and Irving Oyle in holistic medicine.[6]

Case Studies

First case: A thirty-five year old Indian widow with no prior history of depression entered a Yogic institute because of a depressive episode. Her depressive symptoms had increased over the previous ten weeks until she was no longer able to carry out her household responsibilities. She was unable to concentrate, cried spontaneously, had insomnia, and experienced a ten-pound weight loss. Generally she was withdrawn and would stare apathetically. She had three children and lived with her extended family who were helpful and supporative. Her husband had died prematurely one year prior to the onset of her symptoms.

Upon entering a Yogic institute, and "Ashram", she was put on a Yogic breathing regimen. In addition she continued her vegetarian diet and began a supportive relationship with the Yogi and the Ashram milieu. Simple Pranayama consisted in her sitting with legs crossed and with spine erect. She placed her right hand on her left in a supine position in her lap. She was instructed to exhale normally, pause for a few seconds and inhale normally. Exhalation equal to inhalation. She was to look at her own palms and breathe in and out for ten cycles, gradually increasing to ten minute sessions twice daily. After twelve weeks of this treatment she had remission of her symptoms and returned to her family. At a two year follow-up she was symptom-free and functioning well.[7]

Were this same lady seen in a medical clinic she would undoubtedly have been treated with a tricyclic antidepressant for her reactive depression. Very likely her course of recovery would have been similar. Obviously, such an anectodotal report does not control for the variables of Pranayama, the supportive relationships of the Yogi and the milieu, the change in the environment, or the natural course of the illness. The observers believe that Pranayama was a critical ingredient. As with many treatment procedures it should be applied within supportive, culturally acceptable context.

Current neurochemical hypotheses of depression relate it to decreased amounts of norepinephrine at certain CNS neurnonal junctions. Brain serationin levels may be increased or decreased depending whether the depression is agitated or retarded.[8] It is interesting to consider that similar neurochemical changes could have resulted from a controlled breathing regimen. Affective and biochemical changes have been noted to accompany meditative states,[9] but as yet there is a relatively small data base of which procedures to prescribe for specific illnesses.

Second Case.

A sixteen year old Indian male with an eight year history of moderately severe asthma presented himself for Yogic treatment. He had also been treated medically and maintained on aminophylline and cortisone with occassional hospitalizations since age eight. In response to stressful situations at home and in school he would experience asthma attacks. He entered an Ashram and began a program of yogic postures and breathing while remaining in school. Initially Pranayama was introduced in such a manner as to equalize his inhalation and exhalation, gradually progressing to mildly prolonged exhalation. He was taught relaxation exercises and to apply them and controlled breathing when anxious and upset. After six months he had significantly improved and functioned well at school and at home. His medications were put on prn basis. Since he was able to cope with anxiety provoking situations without asthma attacks, his self esteem increase considerably. He continued daily Yogic postures and Pranayama regimen.

Discussion:

Recently Yorkston et al[10] reported improved lung function in bronchial asthma using behavior modification. They noted improvement in patients receiving steroids and undergoing desenitization versus those not receiving steroids or using relaxation alone. Prior studies evaluated symptoms, however this is the first study to reveal a change approaching significance in symptom rating and drug use that corresponds with objective evidence of improved lung function. Although not employing the same methodological approach, it is interesting to note similar psychophysiological improvements in this and other similar cases.[11] As a specific technique, diaphragmatic breathing has been recommended as one approach for several psychophysiologic sequelae of anxiety-related hyperventilation.[12]

Conclusion

Pranayama seems to create a biofeedback-like control of one's physiology. Until recently it was thought impossible to condition the autonomic nervous system, smooth muscles, and glands by other than Pavlovian techniques. Since 1960 there have been reports of learned control with instrumental condtions of a wide range of autonomic nervous system responses including the GSR, heart rate, blood pressure, vasomotor responses, salivation, and the relaxation of striated muscles.[13] Yoga appears to be another option for attaining such physiologic control.[14] Biofeedback and meditative states have also been investigated in the treatment of psychiatric illnesses.[15] Perhaps our Western counterpart of Pranayama is biofeedback which brings with it many innovative approaches to treatment of stress-related disorders.

As an adjunct to standard medical practice, Pranayama and other Yogic procedures may warrant further investigation in emotional problems and in certain psychophysiologic disorders.

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Chronic Illness Treatment and the Self-Concept

Part I

Donna Lou Udelman, M.A.

We are pleased to be a part of this issue honoring our esteemed colleague, respected mentor, and dear friend who has supported our efforts in clinical research, therapy, writing, and presentations.

Chronic illness and its treatment represent particular types of stress. Their impact on patients is determined by such factors as psychological genetic exposure to illness, hereditary predisposition, sex and role definitions, as well as existing and preexisting support systems. I will attempt to address the unique and not-so-unique problems problems faced by women with chronic illness. I will also examine reaction patterns of their families.

All illness activates psychological responses. Regression is one such adaptive response. Healthy regression allows for participation in treatment. "Some patients tend to regress and emphasize the helpless aspects of their illness; they become childish and silly, and in general adopt a 'sick role'. This 'flight into illness' tends to anger both the health-care professional and the family of the patient; thereby further aggravating the already tenuous emotional status of the patient."[1] Both illness and regression leave a patient more vulnerable than before. Conflict arises between independence and dependence. A genuine fear of loss of control relates to the inability to make the illness "go away." Both self-esteem and body-image are involved, with reactions on men and women differing in some aspects.

Men have been taught to be strong. They could not cry. The roles of breadwinner, competitor, and manager have also reinforced the need for health. (In our current culture, "many of the organizations and interests that used to give support to men no longer play any significant role." Thus, "men today have fewer acceptable emotional outlets."[2] The resulting "shell of bravado" is dependent upon sexual and vocational sources of gratification.) Illness, whether acute or chronic, threatens this basic role of a male. Denial can lead to more severe illness, with more serious regression as an outgrowth.

In prior years, most women were involved in care for the home and children. Organization of this area depended greatly on the woman's capacity to integrate the family unit's needs and to set up priorities. The rest of the family was expected to help as required. Expectations of role fulfillment demanded performance of most household responsibilities by the women. Illness, thus, had a significant impact on the functional capacity of the woman at home. If signs or symptoms of disease could easily be discerned, the family could be supportive. If disease was limited to intangibles, such as pain and fatigue, the family's responses might evolve into criticism and avoidance.

With increasing numbers of women working today, the impact of illness on jobs has risen. Whether the woman is working to supplement family income, or to support her family as the primary breadwinner, the need to sustain employment is significant. Very practical issues determine the need to continue working, ie, standard of living, basic needs, and family stability. Women must work hard to attain responsible positions today. Therefore, self-esteem is threatened in many areas when illness intervenes. Because of illness, a woman may fear losses of: identity as a productive worker; role as a family provider; independence; a sense of attractiveness; and the additional family role of wife-mother.

Chronic illness such as arthritis has an evolutionary impact on all facets of a patient's life. Concurrently, it affects the families of patients in many ways. Changes

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From: 333 West Thomas Road, Phoenix, AZ 85013. Presented at the American Psychiatric Association Meeting, Chicago, Illinois, May 16, 1979.

wrought by disease process, include both psychological and physical. The former range from independence to dependence, denial to resignation, and overcompensation to acceptance. Physical changes emcompass limitation of function, loss of attractiveness, and potentially severe crippling. Two concepts are relevant here. The self-concept involves how a patient views himself or herself as a total person. This may involve role, status, goals, and value systems. Body image relates to observable physical and physiological modifications that are products of illness. Thus, an individual with rheumatoid arthritis may face the dual trauma of loss of function and physical attractiveness through the destruciton of varied joints. An additional insult to the integrity of the personality and body-image, may be found in the severity of side effects of medications used to treat the disease itself. The reactions of "significant others" to this insoluble dilemma further complicate the emotional status of patients. To take the option of avoiding treatment, runs the risk of accelerating deterioration. The alternative may involve potential side effects which are equally unacceptable.

The family in chronic illness evolves from the family in health. Change occurs despite or because of homeostatic mechanisms within the family unit. Thus, growth of children, changing status of parents, financial gains and losses, geographic moves, and new careers also modify family reaction patterns. Minuchin[3] "describes family structure as the invisible set of functional demands that organizes the ways in which family members interact. Repeated transactions establish patterns of how, when, to whom to relate, and these patterns underpin the system." Each family member's behavior affects others and "stress in an individual member reverberates strongly" across the entire family system and sub-systems.[4]

Families with good adjustments generally show: 1) clear separation of generational communication; 2) flexibility allowing role shifting as needed; 3) tolerance for individuation; and 4) direct, consistent communication, that maintains self-esteem."[5]

Serious illness requires a reorganization of family following the initial response of denial, anxiety and resentment. "The reorganized family may function as well as or perhaps better than it did before the crisis, or in the process of reorganization family roles and rules may change significantly", with resulting serious emotional pain or impairment of other family members. The family's reorganizaiton may affect the patient's illness and recovery.[6]

The family of a patient with cancer may insist on avoiding discussion of the illness with the patient, protesting that they are protecting the patient. The dynamic explanation of such behavior may lie in their own wish to avoid or deny the painful circumstance. Thus, they may limit the patient's effective discharge of emotions relative to his or her perception of illness. An angry withdrawal may distort the seemingly good intentions leading to strains in current communications and guilt at later times.

My experience has been primarily with patients and families with rheumatologic disorders. Udelman and Udelman[7] have described group therapy with rheumatoid arthritic patients. I have also worked with couples in which one member is an arthritic patient. The impact of arthritis on a patient's spouse and children has been explored, with guilt and repressed rage being common to all members of the family.

Hysterectomy and mastectomy represent two key areas of women and illness. These surgical procedures may be related to benign or malignant disease. Both have special meaning for women. In hysterectomy, the child-bearing capacity is irrevocably lost. If this role has been central to a woman's identity, the surgical procedure may present serious psychological effects. Depression, loss of identity as a woman, and regression may ensue. If the hysterectomy has been done because of malignancy, the patient's anxieties over a life-threatening illness may be relieved dramatically. The concern over pregnancy is prominent for some women. Hysterectomy, then, relieves such fears.

Mastectomy generally is performed to remove malignant lesions. It has significant meaning because of the threat both to narcissism and life. Self-image and body-image are affected. Loss of body part requires working through grief. Family support is critical in this process. Relief in the removal of a life-threatening lesion must be accompanied by re-integration of feminine identity. Prostheses are helpful in this area. Psychotherapy may be helpful to overcome the emotional crisis.

Disruption of health in any family member produces a ripple effect throughout the family. Homeostasis is interrupted, and major efforts are necessary to reestablish a new functional level. Education and support may assist patients and their families in many illnesses today. Training programs, such as at the Phoenix Arthritis Center, have been utilized to increase familiarity with illness and treatment. Varied therapeutic approaches such as conjoint, family individual and group may be necessary. The family in chronic illness is a family in need of support.

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Chronic Illness Treatment and the Self-Concept

Part II

Harold D. Udelman, M.D.

Medicine generally treats illness in an ongoing manner, designed to "cure" when possible; relieve often; and support and counsel always. Few illnesses are subject to total cures. Many remit with or without intervention. Most require management to modify course, produce remissions, and enable patients to function as well as possible.

Today's concerns dictate attention to prevention of illness and control of costs. Both of these are of great importance to society as a whole and to each of us, individually. Prevention is an evolving concept which has brought great strides in many areas. It has, additionally, wrought much confusion in the multitude of restrictions implied or intended on diet, activity, and such exotic pursuits as respiration. Cost control has focused on relative values in care systems. The outgrowth of such concerns could lead to apportionment of available resources, with consequent scarcity of the "medical commodity." While prevention research continues, health care specialists must treat illness, much of which is chronic—much of which is costly in its long term impact.

The prevention of chronicity has been discussed in a recent article by Ibrahim S. Turek.[1] The goal of prevention is important—the achievement of such a goal remains elusive.

Chronic illness, whether physical or emotional, produces changes within the patient and his or her environment. Unipolar or bipolar depression has significant impact on the self-image. Schizophrenia can also grossly distort self-image. In remissions, pragmatic, objective views can be regained. With continued exacerbations, however, the ego suffers further insults, intensifying the conflict over self-concept. Disturbance of function, impaired independence and loss of hope may be outgrowths.

Physical illness disrupts adaptational patterns in similar ways. Independent, responsible goal-oriented individuals are faced with the need for regression to convalesce. When illness is chronic, repeated regressions remain an ever-looming threat. Hope is severely affected. Various physical illnesses produce different responses by patients and families. In chronic lung disease—emphysema—the gradual loss of adequate pulmonary function limits not only routine activity, but also threatens existence. Pattison, Rhodes, and Dudley[2] discussed a group therapy program with inpatients who had chronic lung disease. Arousal of affect was found to be adverse in its impact, to the point of being potentially life-threatening. Self-concept is obviously impaired by the severe, progressive nature of the illness.

Rheumatoid arthritis is a chronic whimsical illness which waxes and wanes. It is a multi-system disease wherein gastrointestinal and cardiovascular complications may worsen the real and perceived severity of illness. Ophthamologic effects can also be found. Primarily, though, this disease ravages joints. In

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From: 333 West Thomas Road, Phoenix, AZ 85013. Presented at the American Psychiatric Association Meeting Chicago, Illinois, May 16, 1979.

some cases, deformities and crippling severely affect function and self-concept. Loss of attractiveness, strength, and self-esteem disrupts the patients's life. It also changes the family constellation in many ways. Spouses may overcompensate or abandon; children may rebel against or succumb to added pressures; and patients may lapse into stoicism or evolve into "benevolent tyrants." Udelman and Udelman[3] have worked with individuals, couples and families who are reacting to this chronic illness.

Cancer represents another form of chronic illness that may also encompass aspects of both acute and subacute states. The self-concept is directly threatened by the diagnosis—with potential for fatality clearly a concern. Where prior years found no treatment and/or "cure" for cancer, expectations today embody percentages of survival, modalities of treatment, and longevity statistics. Chronicity may follow "successful" treatment—but this may produce new problems for the self-concept, weakened by illness and uncertain because of prognosis.

A working definition of self-concept would be that system of roles, functions and responsibilities assumed by the individual; it would relate to the body-image and sense of attractiveness felt; and it would include elements of success and/or failure in performance. A health self-concept enhances function—it is guided by goals linked to an ego-ideal.

Treatment of chronic illness involves instruction, restriction, pharmacotherapy, surgical intervention, and physiotherapy. Various illnesses require additional modalities such as radiation and inhalation therapy. Use of potent pharmacologic agents requires monitoring of physical and laboratory findings. Often, it may not include monitoring emotional findings.

The patient who receives adequate instruction on the primary and secondary effects of medication is likely to be the patient who makes an informed choice to participate in his or her treatment. Family instruction also is invaluable in promoting such compliance.

Patients are often confronted by dilemmas of treatment. The potency of certain medications carries with it, the probability of side effects. In psychiatry, we have found increasingly that we cannot treat chronic illness without the potential for side effects. Examples here include the emergence of Tardive Dyskinesia with major tranquilizer usage, cardiac effects of tricyclic anti-depressants, or the toxic effects potential in carefully monitored lithium therapy. The self-concept is clearly involved when patients develop Tardive Dyskinesia, cardiac irregularities, or neurological sequelae. To refuse treatment is to risk an emotionally damaged self-concept; to accept, is to risk untoward side effects. Thankfully, these issues are not as frequent as in many physical illnesses.

The use of steroids in rheumatoid arthritis indicates more severe illness. Thus, patients who receive such medications begin with a defined adverse status. Side effects further complicate treatment and self-concept. Weight gain, moon-shaped facies, striae, euphoria, and psychosis are all possible. Attractiveness and body-image are distorted; dependency is increased; and hope lessened. Engle's[4] concept of hopelessness and helplessness is relevant here.

Treatment with other potent medications such as remission inducers and immunosuppressives pose problems of potentially severe side effects that include susceptibility to infection, ophthalmologic effects, and renal damage. The physician's "Hobson's choice" matches the patient's then. Therapeutic intervention runs risks that may severely alter physical well-being as well as self-concept in either adverse or salutary ways.

Treatment of cancer further clarifies the impact of chronic illness, treatment, and the self-concept. Surgical procedures may grossly distort the self-concept, while preserving life. Quality of life is basic in the choice of treatment modality. Radiation therapy, again, may preserve life, while altering its pattern irrevocably. Finally, chemotherapy has produced many notable changes in the field of oncology. These potent drugs—which are also utilized in other fields such as rheumatology—may produce nausea, vomiting, agranulocytosis, and other side effects.

It would be folly to dismiss treatment only as a source of damaged self-concepts. To the contrary, without the hope for preservation of life, there can be no hope for functional self-concept. Maintenance of function is an essential goal of treatment. It enhances the recovery of patients, and contributes to an acceptably re-integrated ego. Though this self-concept may be organized at lesser level because of illness and/or treatment, it nonetheless represents a "healthy" accomodation to these stresses.

In summary, it becomes apparent that medicine must always seek new, more effective means of combatting illness; that these new means both relieve and complicate emotional recovery; and that members of the helping professions must monitor, treat and support the self-concept as well as the symptoms. The late Maurice Levine, M.D., my highly respected mentor, used to comment on the continuing need in medicine for action on the basis of insufficient evidence. Nowhere is this more evident than in the continuing effort to prevent deterioration, treat exacerbation, and support emotionally those patients in whom we diagnose chronic illness.

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Editorials

Therapeutic Impact of a Modern Amputation Program

As of 1976, about one in 300 Americans have had a major extremity amputation. There are approximately 30,000 new amputations performed in the United States each year. Nearly two-thirds of these amputations (17,850) are lower extremity amputations performed for complications of diabetes mellitis and/or atherosclerotic vascular disease. Approximately 12,000 upper extremity amputations are performed each year. The most common cause of upper extremity amputation is accidents. Most lower extremity amputations are performed in patients over 55 years of age and, therefore, lower extremity amputation has only a moderate impact on the working segment of our society. However, more than 75% of the upper extremity amputations are performed on male patients less than 55 years of age and, therefore, upper extremity amputations have a major impact on the working segment of our society, especially in highly industrialized areas where accidents resulting in loss of a limb or part of a limb are relatively common.

A review of national data demonstrates that after lower extremity amputation only 50% of the patients become rehabilitated and regain independent function. The average rehabilitation time after lower extremity amputation is in excess of four months (mean 135 days). A similar review of national data for patients undergoing upper extremity amputations demonstrates that, at best, only 50% of the patients become functional. The average rehabilitation time after upper extremity amputation is about six months (mean 180 days).

In most medical settings, amputations are performed infrequently and sporadically. In such an environment, it is extremely difficult for an individual medical practitioner to present a prospective amputation patient with both a coordinated surgical and rehabilitation program. At the University of Arizona/Tucson Veterans Administration Medical Center, and Amputation Rehabilitation Program has been established which is capable of providing patients with a team approach to amputation, postamputation rehabilitation, and longitudinal follow-up for both medical and prosthetic care. In addition to the team approach, the Amputation Rehabilitation Program uses advanced surgical and prosthetic techniques for both upper and lower extremity amputations. These combined efforts provide a unique investigative and educational area in addition to the health care benefits.

Approximately ten years ago, we began evaluating immediate postoperative prosthetic fitting for lower extremity amputee patients. This program not only allowed the construction of a temporary prosthetic limb at the time of surgery, but also allowed the amputation patient to begin rehabilitation and ambulation on the first day after surgery. We have recently extended the immediate postoperative prosthetic fitting concept to include upper extremity amputation patients. Perhaps the most unusual aspect of our current program is the inclusion of electric hands, electric elbows and myoelectric "bionic" hands into the immediate postoperative fitting process so that a patient with an upper extremity amputation awakens from surgery with a functional hand and arm.

Implementation of these techniques at the University of Arizona/Tucson Veterans Administration Medical Center has resulted in greater than 95% rehabilitation within 30 days for lower extremity amputation (compared to the 60% rehabilitation in 135 days for conventional treatment) and 100% rehabilitation within two weeks for upper extremity amputation (compared to the 60% rehabilitation in six months). In addition to improved rehabilitation rates, markedly improved rehabilitation time, and decreased surgical morbidity and mortality, this type of amputation program is extremely cost-effective. A study done within the V.A. suggests that this program, if applied on a national scale, is capable of saving $80,000,000 over five years within the V.A. Hospital system. A similar analysis within the city of Tucson suggests that utilization of the University of Arizona/Tucson Veterans Administration Medcial Center Amputation Rehabilitation Program as a city-wide project would save an estimated $500,000 per year in health care costs for amputee patients.

An additional counterpart to the amputation program is the co-existence of a limb reimplantation team as part of the Amputation Rehabilitation Program. If a patient presents to either the University of Arizona or the V.A. Medical Center with an amputated extremity and the amputated part, we have both the technical capabilities and expertise to reimplant the patient's finger, hand, arm, or leg, if appropriate, rather than complete the amputation. If, however, the limb reimplantation team feels that limb reimplantation will not result in good extremity function, we also have the opportunity of including such a patient in our immediate postoperative prosthetic program with full knowledge that the patient will achieve rapid rehabilitation and become a functional member of society.

The Amputation Rehabilitation Program at the University of Arizona/Tucson V.A. Medical Center is unique not only to Tucson, but also to the state of Arizona. The program provides enormous patient benefits and is extremely cost-effective. Utilization of both helicopter and fixed-wing aircraft allows patients to be transported to either the University or the V.A. facility from any point within the state of Arizona. Based upon patient benefits, proven quality of medical care, and cost-effectiveness, it would seem reasonable that this program should receive the hearty endorsement of both physicians and patients alike.

James M. Malone, M.D.
Assistant Professor Surgery
Assistant Chief/Vascular Surgery
Louis J. Kettel, M.D.
Dean, College of Medicine

Auxiliary Highlights

September Conference

Purpose, planning and perseverance lead to productive living — and that's the theme for Arizona Auxiliary's 1979 Fall Conference, September 24-25, at the ArMA offices.

The conference is open to all Auxiliary members. It's of special importance for county and state officers and board members. Once again, the goal is to educate ourselves to be more effective volunteers. Leadership training is a vital part of the Auxiliary program at county, state and national levels.

Guest speakers will be Marty Squires, who has served as a faculty member for the American Hospital Association Institutes for Auxilians, and Emily Kimball, who has an M.A. in personnel management and is chairman of the Phoenix Junior League training Committee. Both women have many years of experience as volunteers in a variety of community organizations and both are known for their expertise in training volunteer boards.

Marty Squires will speak at 7:30 Monday evening on "Body Language and Communication". ArMA members are invited to attend this presentation and the 6 p.m. supper which precedes it.

Thursday morning, Emily Kimball will lead a workshop on "Management by Ojective". Following a light luncheon, served by Auxiliary hostesses, she will speak at 1 p.m. on "Time Management".

Other conference speakers include Joyce Newman, ArMA Auxiliary president-elect; Connie Wry, Pima County Auxiliary president who will discuss "The Case of the New President", Nancy Henderson, Auxiliary parliamentarian; and Sue Gigax, who will introduce the new national health education project "Shape Up for Life".

Joyce Newman planned this year's conference with the assistance of Judy Davis of Prescott, second vice president. The Auxiliary is grateful to ArMA for making its facilities available to us for this meeting and to the ArMA staff for assistance in arrangments.

National Convention Report

Arizona's seven delegates to the AMA Auxiliary Convention in Chicago, July 20-25, were kept busy. They—

*Attended eight hours of workshops on AMA-ERF, Membership, Health Projects and Legislation.

*Watched as an Auxiliary check for more than one million dollars was presented to the AMA's Education and Research Fund. This money was raised by county and state Auxiliaries in a myriad of ways.

*Took part in the spirited election of a president-elect, which saw our candidate, Mrs. John Vaughan of Washington, defeat Mrs. Hoyt Gardner of Kentucky, who had been nominated from the floor.

*Cheered as Arizona's Ella Kimball of Phoenix won re-election as a director on the national board and was also elected to the nominating committee.

*Were outvoted as the House of Delegates approved a national dues increase of $4.00 in 1980 and another $4.00 in 1982. The Arizona delegation supported an amendment which would have limited the increase to $4.00 in 1980.

*Rejoiced at receiving an award for the highest increase in membership of the states in the Western Region.

Arizona was represented by Joyce Newman, Janet Hoffmann, Trudie Anand, Jeanne Weiland, Sue Gigax, Mary Eleanor Holmes and Eloise Clymer.

National Confluence

October 7-9 are the dates for the Auxiliary Fall Confluence in Chicago. Presidents-elect of Arizona's six organized county Auxiliaries will go to Chicago with the state president and president-elect for a three-day briefing on new health projects and current legislation. The meeting will provide plenty of opportunities for us to exchange ideas with leaders of other Auxiliaries.

Transportation for county officers will be paid by the AMA Auxiliary, if the spouse is a member of AMA. The state Auxiliary will pay for hotel rooms and registration fees, which include all working meals. Another example of how Auxiliary dues—state and national—finance training for leadership in the counties!

Mrs. John T. Clymer,
ArMA Auxiliary President

President
William E. Crisp, M.D.

President Elect
John T. Clymer, M.D.

Vice President
Clyde W. Kurtz, M.D.

Secretary
John A. Ash, M.D.

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Walter V. Edwards, M.D.
C. Herbert Fredell, M.D.
Benjamin K. Harris, M.D.
John W. Kennedy, M.D.
William B. McGrath, M.D.
John Stoner, III, M.D.
William C. Weese, M.D.
Donald J. Ziehm, M.D.

ArMA Reports

THE MINUTES APPEARING IN THIS SECTION HAVE BEEN EDITED TO CONSERVE SPACE. A COMPLETE COPY OF THE MINUTES OF ANY MEETING WILL BE MAILED TO ANY MEMBER REQUESTING THEM.

PROFESSIONAL COMMITTEE

Meeting of the Professional Committee of the Arizona Medical Association, Inc., held at 810 W. Bethany Home Road, Phoenix, Arizona, on Tuesday, July 10, 1979, convened at 7:01 p.m., Joseph W. Hanss, M.D., Chairman, presiding.

Welcome

The Chairman welcomed Donald B. Kunkel, M.D. and James B. Scott, M.D. as new members of the Committee.

Section on Aging and General Medicine

Biofeed Back

The Committee note the April 25, 1979, action of the Board of Directors to table the recommendations of the Professional Committee made during its meeting of 4/3/79 relative to biofeedback and advertising. The Committee received a letter from Samaritan Health Service apologizing for any possible problems the enthusiastic marketing of their Samaritan Stress Center may have caused the Association. RECIEVED FOR INFORMATION.

Section on Allied Medical Groups

Podiatry

Dr. Dupont, the Section Chairman, informed the Committee that meetings between the orthopedic surgeons and the podiatrists relative to mandating hospital privileges for podiatrists have ceased. There is presently talk of the podiatrists filing suit against hospitals, hospital staffs and hospital staff committees to gain privileges without legislative activity. There was considerable discussion. RECEIVED FOR INFORMATION.

Legislation Activity

The Joint Select Committee on Allied Health Professions and Other Regulatory Commissions has had one meeting for organizational purposes. Two other meetings of this Committee have been scheduled. RECEIVED FOR INFORMATION.

Nurse Practitioners

The Committee receive minutes from the June 19, 1979, meeting of the Phoenix Obstetrical and Gynecological Society Subcommittee on Nurse Practitioners. It was noted that the nurse practitioners would be again introducing legislation to broaden their scope of practice and take control of nurse practitioners from the Joint Board of Nursing and Medical Examiners. There was general discussion. RECEIVED FOR INFORMATION.

Homoeopathy

The Committee received a report from the Association's public relations firm on the Homoeopathic College of Medicine Open House as well as a news release about the Homoeopathic College opening in Phoenix in August. It was noted that during the last legislative session a bill was introduced which would have established a licensing board of homoeopathic physicians; this legislation did not pass but will most likely be re-introduced during the next session. There was discussion.

IT WAS MOVED AND CARRIED TO REFER THE MATERIAL ON THE HOMOEOPATHIC COLLEGE OF MEDICINE OPENING IN PHOENIX IN AUGUST, 1979, TO THE BOARD OF DIRECTORS FOR THEIR INFORMATION AND POSSIBLE ACTION.

Section on Cancer

Dr. Hanss informed the Committee that the Chairmanshiop of the Section on Cancer is vacant. The bylaws do not specify that the Professional Committee comprise a Section on Cancer. There was discussion on the alternatives open for either appointing a new Chairman or dropping the Section.

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE BOARD OF DIRECTORS THAT THE SECTION ON CANCER OF THE PROFESSIONAL COMMITTEE BE DROPPED AS A STANDING SECTION OF THE PROFESSIONAL COMMITTEE. (This does not require a bylaw amendment.)

Section on Emergency Care & Disaster Medicine

Free Standing Emergency Facilities

The Committee noted the action of the Board of Directors in their meeting held April 25, 1979, to endorse the standards for minimal requirements for free standing emergency facilities adopted by the Arizona Chapter, American College of Emergency Physicians in September, 1978. It was noted that Arizona is one of only three states who have taken strong positive action regarding minimum standards for free standing emergency facilities. RECEIVED FOR INFORMATION.

The Committee reviewed a news release announcing the opening of an emergency center in East Mesa. It was noted that, from the information contained in the news release, this center does not meet the minimal requirements for free standing emergency facilities endorsed by ArMA. There was considerable discussion and the membership concurred that additional data on free standing emergency facilities is necessary before any action on implementing the guidelines could be taken.

IT WAS MOVED AND CARRIED TO DIRECT STAFF TO COLLECT DATA ON FEE STANDING EMERGENCY FACILITIES IN ORDER TO BEST DETERMINE HOW TO IMPLEMENT THE ArMA/ACEP GUIDELINES.

Section on Health Education

Dr. Niccum reported that he attended a health education symposium sponsored by the Red Cross. Although many interesting topics were discussed, there has been no follow through by the attendees. Dr. Niccum stated that this section could be very active, however, he will need assistance. He is actively seeking interested physicians to serve on this section. RECEIVED FOR INFORMATION.

Section on Poison Control

Dr. Kunkel informed the Committee of the legislative activitiy with regard to establishing a statwide poison control center and that the bill was vetoed (as part of the certificate of need bill). There is a need for a combined approach from all areas of the state to come up with a comprehensive program for poison control. Legislation is likely to be introduced during the next session for the establishement of a poision control center in Arizona. RECEIVED FOR INFORMATION.

Section on Sports Medicine

Annual Sports Medicine Symposium

Dr. Brainard announed that his section is formulating the plans for the 6th Annual Sports Medicine Symposium. ArMA will broaden its alliance with the ancillary organizations in the sponsorship of the program. There will be efforts to proivde a more basic educational program this year. RECEIVED FOR INFORMATION.

Action on Smoking and Health

The Association has been asked by "Action on Smoking and Health" (A.S.H.) to endorse or co-sponsor a public meeting on October 5, 1979, in Phoenix vicinity, to seriously analyze the public health issues which relate to smoking. Dr. Brainard added that Frank Kush would be honored at the meeting for his work against smoking in the athletic realm. The U of A College of Medicine, the Indian Health Service, leading teaching hospitals of the state and leading voluntary oganizations have already given their endorsement.

IT WAS MOVED AND CARRIED TO RECOMMEND TO THE BOARD OF DIRECTORS TO ENDORSE THE "ACTION ON SMOKING AND HEALTH" COMMITTEE MEETING TO BE HELD ON OCTOBER 5. 1979, TO ANALYZE THE PUBLIC HEALTH ISSUES WHICH RELATE TO SMOKING AND TO HONOR FRANK KUSH FOR HIS INVOLVEMENT.

PUBLIC RELATIONS COMMITTEE

The meeting of the Public Relations Committee of the Arizona Medical Association, Inc. held at 810 W. Bethany Home Road, Phoenix, Arizona, on Thursday, August 2, 1979, convened at 7:03 p.m., Ira B. Ehrlich, M.D., Chairman, presiding.

Florida Medical Association Flims

Graham B. Kretchman, M.D. addressed the committee, prefacing the viewing of two films obtained from the Florida Medical Association, briefly stating the reasoning behind the making and distribution of the films, the reactions of certain Phoenix groups who has already viewed the films and the possibilities of a like project being considered by the committee in relation to it's primary function, i.e., improving the image of the medical profession in the eyes of the general public.

After viewing the film, "A Matter of Life," and the video tape, "It's Your Life," it was the consensus of the membership that, while both were well done, the context of the video tape would be far better received by the lay person. The committee felt that, in order for it to make any final determinations regarding the possibility of pursuing a similar project by the Arizona Medical Association, either by itself or in conjunction with other state medical organizations, it would be necessary to obtain from the Florida Medical Association more in-depth information regarding cost of production, the initial intent for and usage of both the film and video tape, the number of and responses from groups having used the film and/or tape prior to this time.

It was agreed that, when all such information has been assembled, a meeting of the committee would be called to present to the entire membership all of the facts in order that the committee could make any final recommendations concerning the feasibility of such a project.

Other Business

Myopractics

The committee received and discussed a news release regarding a press conference to be held at 9:30 a.m., on Friday, August 3, 1979, concerning the recent decision of the Superior Court that one Jym Marinakis, Myrorractor, is practicing "some form of medicine" without a license.

EXECUTIVE COMMITTEE

The meeting of the Executive Committee of the Arizona Medical Association, Inc. held at 810 West Bethany Home Road, Phoenix, Arizona, on Wednesday, July 11, 1979, a quorum being present, convened at 6:37 p.m., William E. Crisp, M.D., President and Chairman, presiding.

1979 AMA Annaul Meeting - Review of Delegation Handbook

The committee and members of the delegation who were present reviewed the handbooks previously distributed to the delegation in preparation for their attendance at the 1979 AMA Annual Meeting to be held in Chicago later this month, discussing at length those matters which were of immediate concern to the Association. Additionally, the delegation addressed the committee regarding the two resolutions it planned to introduce, i.e., Nos. 54 and 145, relating to PL 93-641; Composition of HSA Board of Directors and State Medical Associations as Accrediting Bodies, respectively. Also presented for review and discussion was the Final Report of the AMA Ad Hoc Committee on Principles of Medical Ethics.

Correspondence

ICA Hearing/Medical Economics Committee

A letter received from Richard W. Switzer, M.D. regarding the recent hearings of the Industrical Commission of Arizona and setting forth positions taken by the Medical Economics Committee and Dr. Switzer was reviewed by the committee and it was the consensus that Dr. Switzer should be invited to attend the next meeting of the Executive Committee in order for there to be a complete and full discussion regarding the matter.

Other Business

Action on Smoking and Health

The committee considered a recent request for Association support of a program being co-sponsored by A.S.H. (Action on Smoking and Health), the University of Arizona, the Indian Health service, and others to be held in the Phoenix area on October 5, 1979. Following discussion.

IT WAS MOVED AND CARRIED THAT THE ARIZONA MEDICAL ASSOCIATION SUPPORT THE PROGRAM TO BE HELD IN THE PHOENIX AREA ON OCTOBER 5, 1979, BY A.S.H. (ACTION ON SMOKING AND HEALTH), THE UNIVERSITY OF ARIZONA, THE INDIAN HEALTH SERVICE, AND OTHERS FOR THE PURPOSE OF SERIOUSLY ANALYZING PUBLIC HEALTH ISSUES RELATIVE TO SMOKING.

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Manager of Valley Bank's
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Chuck and his staff give you the dependable and complete service professionals always expect from Valley Bank. The bank that's helped thousands of doctors meet their specialized needs over the years.

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Ad Hoc Committee on Review Committees.

The minutes of the June 28, 1979 meeting of the newly-formed Ad Hoc Committee on Review Committees, together with proposed revised guidelines for the Underwriting and Claim Review Committees promulgated therby were reviewed and considered by the executive committee and it was the consensus that the same be submitted to the Mutual Insurance Comapny of Arizona for its review, following which a conference committee meeting would be held to finalize the matters presently at issue.

Florida Medical Association

The committee was advised that a request had been received from Graham B. Kretchman, M.D. regarding the viewing of certain flims available through the Florida Medical Association relating to the medical community, its present position on major issues, etc. on the premise of possibly doing a similar project in Arizona for public relations purposes. It was the consensus of the membership that the matter should be referred to the Public Relations Committee for further contact and follow-up as it deemed appropriate.

AMA Jail Proposal

The committee received and reviewed a follow-up from the American Medical Association relative to the project considered by this committee in meeting on June 7, 1979. Following the committee's action at the June 7th meeting, it was determined that no funds could be made available through federal agencies; and, therefore, due to the timing of the request and lack of funding, no participation by the Association could be undertaken at this time.

Tenuate® Ⓒ
(diethylpropion hydrochloride NF)
Tenuate Dospan®
(diethylpropion hydrochloride NF) controlled-release

AVAILABLE ONLY ON PRESCRIPTION
Brief Summary
INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction. The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below
CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma Agitated states Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).
WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect, rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle, the patient should therefore be cautioned accordingly *Drug Dependence* Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program. Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia. *Use in Pregnancy* Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children*: Tenuate is not recommended for use in children under 12 years of age.
PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias. Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary
ADVERSE REACTIONS: *Cardiovascular* Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System*: Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache; rarely psychotic episodes at recommended doses. In a few epileptics an increase in convulsive episodes has been reported *Gastrointestinal*: Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic*: Urticaria, rash, ecchymosis, erythema, *Endocrine*. Impotence, changes in libido, gynecomastia, menstrual upset *Hematopoietic System*: Bone marrow depression, agranulocytosis, leukopenia. *Miscellaneous*: A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.
DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in midevening if desired to overcome night hunger. Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg tablet daily, swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.
OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard. Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of April, 1976

MERRELL-NATIONAL LABORATORIES Inc.
Cayey, Puerto Rico OD633

Direct Medical Inquiries to
MERRELL-NATIONAL LABORATORIES
Division of Richardson-Merrell Inc.
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References: 1. Citations available on request from Medical Research Department, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215. **2.** Hoekenga, M.T., O'Dillon [Dillon], R.H., and Leyland, H.M. A comprehensive review of diethylpropion hydrochloride. In, Central Mechanisms of Anorectic Drugs, S Garattini and R. Samanin, Ed., New York, Raven Press, 1978, pp 391-404

Merrell

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Overweight may not always be simple... complications can develop.*

Complicated or not...

Tenuate® Dospan® ⒸIV

(diethylpropion hydrochloride NF)

75 mg. controlled-release tablets

A useful short-term adjunct in an indicated weight loss program.

Overweight patients in certain diagnostic categories often require strict appetite control and a successful program of weight reduction may tend to diminish the incidence or severity of the complications in some patients. Diethylpropion hydrochloride has been reported useful in such patients and while it is not suggested that Tenuate itself in any way reduces the complications of overweight, it may have a useful place as a short-term adjunct in a prescribed dietary regimen. **Tenuate should not be administered to patients with severe hypertension; see additional Warnings and Precautions on the opposite page.**

In uncomplicated overweight.

Many patients, on the other hand, present with excess fat but no disease. While this condition is often termed uncomplicated obesity, complications of both a social and a psychologic nature may be distressingly real for the patients. In these cases, a short-term regimen of Tenuate can help reinforce your dietary counsel during the important early weeks of an indicated weight loss program.

Clinical effectiveness.

The anorectic effectiveness of diethylpropion hydrochloride is well documented. No less than 16 separate double-blind, placebo-controlled studies attest to its usefulness in daily practice.[1] And the unique chemistry of Tenuate provides "...anorectic potency with minimal overt central nervous system or cardiovascular stimulation."[2] Compared with the amphetamines, diethylpropion has minimal potential for abuse.

Tenuate–it makes sense. And it's responsible medicine.

*Studies have shown that obesity is associated with an increased incidence of hypertension, symptomatic heart disease, adult-onset diabetes, and other diseases.

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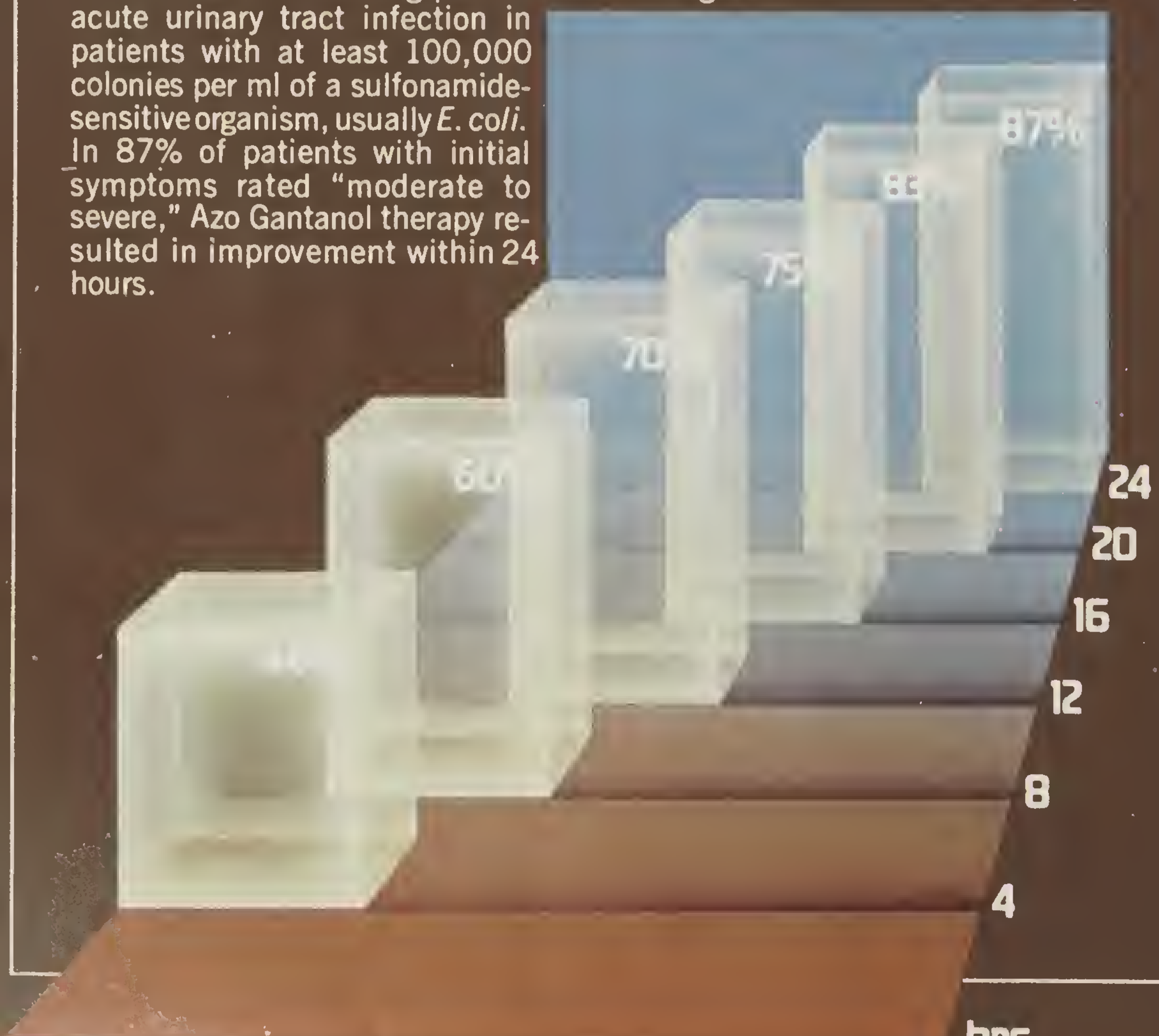
For prescribing information see opposite page

Important data on the pain of acute cystitis:

In 87% of patients studied [303 of 349], Azo Gantanol® reduced pain and/or burning within 24 hours*

A controlled, multicenter study assessed the efficacy of Azo Gantanol in relieving pain and/or burning associated with acute urinary tract infection in patients with at least 100,000 colonies per ml of a sulfonamide-sensitive organism, usually *E. coli*. In 87% of patients with initial symptoms rated "moderate to severe," Azo Gantanol therapy resulted in improvement within 24 hours.



Fast pain relief plus effective antibacterial action

Azo Gantanol®

Each tablet contains 0.5 Gm sulfamethoxazole and 100 mg phenazopyridine HCl.

for the pain for the pathogens

Before prescribing, please consult complete pr uct information, a summary of which follows:

Indications: In adults, urinary tract infections complicated by pain (primarily pyelonephritis, pyelitis and cystitis) due to susceptible organi (usually *E. coli, Klebsiella-Aerobacter, Staphyl coccus aureus, Proteus mirabilis*, and, less frequently, *Proteus vulgaris*) in the absence of obstructive uropathy or foreign bodies. **Note: C fully coordinate *in vitro* sulfonamide sensitivity tests with bacteriologic and clinical response; aminobenzoic acid to follow-up culture media. Increasing frequency of resistant organisms lim the usefulness of antibacterials including sulfonamides.** Measure sulfonamide blood levels variations may occur; 20 mg/100 ml should be maximum total level.

Contraindications: Children below age 12; sulfonamide hypersensitivity; pregnancy at term a during nursing period; because Azo Gantanol c tains phenazopyridine hydrochloride it is cont dicated in glomerulonephritis, severe hepatitis, uremia, and pyelonephritis of pregnancy with disturbances.

Warnings: Safety during pregnancy not establis Deaths from hypersensitivity reactions, agranu tosis, aplastic anemia and other blood dyscrasi have been reported and early clinical signs (so throat, fever, pallor, purpura or jaundice) may i dicate serious blood disorders. Frequent CBC a urinalysis with microscopic examination are re ommended during sulfonamide therapy.

Precautions: Use cautiously in patients with i paired renal or hepatic function, severe allergy, bronchial asthma; in glucose-6-phosphate dehydrogenase-deficient individuals in whom dose-related hemolysis may occur. Maintain adequate fluid intake to prevent crystalluria a stone formation.

Adverse Reactions: *Blood dyscrasias* (agranulocytosis, aplastic anemia, thrombocytopenia, leukopenia, hemolytic anemia, purpura, hypo thrombinemia and methemoglobinemia); *allerg reactions* (erythema multiforme, skin eruptions Stevens-Johnson syndrome, epidermal necroly urticaria, serum sickness, pruritus, exfoliative dermatitis, anaphylactoid reactions, periorbital edema, conjunctival and scleral injection, phot sensitization, arthralgia and allergic myocarditi *G.I. reactions* (nausea, emesis, abdominal pai hepatitis, diarrhea, anorexia, pancreatitis and stomatitis); *CNS reactions* (headache, peripher neuritis, mental depression, convulsions, ataxi hallucinations, tinnitus, vertigo and insomnia); *miscellaneous reactions* (drug fever, chills, toxi nephrosis with oliguria and anuria, periarteritis nodosa and L. E. phenomenon). Due to certain chemical similarities with some goitrogens, diuretics (acetazolamide, thiazides) and oral hyp glycemic agents, sulfonamides have caused ra instances of goiter production, diuresis and hyp glycemia. Cross-sensitivity with these agents exist.

Dosage: Azo Gantanol is intended for the acute painful phase of urinary tract infections. *Usual adult dosage:* 2 Gm (4 tabs) initially, then 1 G (2 tabs) B.I.D. for up to 3 days. If pain persist causes other than infection should be sought. After relief of pain has been obtained, continu treatment with Gantanol (sulfamethoxazole) ma be considered.

NOTE: Patients should be told that the orange- dye (phenazopyridine HCl) will color the urine.

Supplied: Tablets, red, film-coated, each conta ing 0.5 Gm sulfamethoxazole and 100 mg phenazopyridine HCl—bottles of 100 and 500.

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Roche Laboratories
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Nutley, New Jersey 07110

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*Data on file, Hoffmann-La Roche Inc., Nutley, New Jersey 07110.

Future Medical Meetings

CONTINUING MEDICAL EDUCATION

The following institutions and organizations have been accredited for their continuing medical education programs by the Liaison Committee on Continuing Medical Education

Arizona Heart Institute
Arizona Medical Association
Arizona State Hospital, Phoenix
Camelback Hospital, Phoenix
Desert Samaritan Hospital, Mesa
Good Samaritan Hospital, Phoenix
Health Maintenance Associates, Phoenix
Maricopa County General Hospital, Phoenix
Mesa Lutheran Hospital, Mesa
Phoenix Baptist Hospital & Health Center
Phoenix Indian Medical Center
Phoenix Memorial Hospital
Phoenix Psychiatric Council
St. Joseph's Hospital & Medical Center, Phoenix
St. Lukes's Hospital & Medical Center, Phoenix
St.Mary's Hospital, Tucson
Scottsdale Memorial Hospital
Tucson Hospitals Medical Education Program, Tucson
Veterans Administration Hospital, Phoenix
Veterans Administration Hospital, Prescott
University of Arizona College of Medicine, Tucson

The accredited institutions and organizations above produce a variety of continuing medical education programs. Each accredited institution and organization is responsible for designating which of these programs meet ArMA's requirements for Category 1 credit. Physicians who participate in programs which are designated Category 1 by accredited institutions will receive Category 1 credit toward the ArMA Certificate in CME and the AMA's Physician's Recognition Award.

SEPTEMBER

ACLS Recertification Course
September 21, 1979. Arizona Medical Association offices, 810 West Bethany Home Road, Phoenix, AZ 85013. Sponsor: Arizona Affiliate of the American Heart Assoc. Contact: Carol Turner, ArMA Offices, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

OCTOBER

Practical IOL Course
October 6-7, 1979, Minneapolis, MN 55435, Radisson-South, 7800 Normandale Blvd. Contact: Samuel L. Pallin, M.D., 10503 Thunderbird Blvd. Ste 9, Sun City, Az 85351. Sponsor: Surgidev Corporation.

West Coast Society of Allergy and Immunology, 18th Annual Scientific Session
October 11-13, 1979. MGM Grand, Las Vegas, Nevada. Sponsor: West Coast Society of Allergy & Immunology. Contact: Daniel H. Goodman, M.D., Program Chairman, West Coast Society of Allergy and Immunology, 5210 S.W. Corbett Ave., Portland, Oregon 97201. Approved for 15 hours of category 1 credit toward the AMA's Physicians Recognition Award.

Second Annual Symposium on Lifestyles and Health
October 13-141, 1979. Sherton-Place Hotel, San Francisco. Sponsor: Unov. of California San Francisco Schools of Medicine, Nursing, Denistry and Pharmacy. Contact: Continuing Education Helath Sciences, 24 Kirkham, San Francisco, CA 94143. CME credit available.

ACLS Provider Course
October 19 & 20, 1979. Arizona Medical Association offices, 810 W. Bethany Home Road, Phoenix, AZ 85013. Sponsor: Arizona Affiliate of the American Heart Assoc. Contact: Carol Turner, ArMA Offices 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

Eighth Annual Symposium of the Western Division of the American Geriatrics Society. Management of Vascular Disease In the Elderly
October 26-27, 1979. Lakes Club, Sun City, Arizona. Sponsor: Univ. of Arizona College of Medicine and Walter O. Boswell Memorial Hospital. Contact: Julius Schwimmer M.D., 13220 105th Ave., Bldg. B, Rm 61, Sun City, AZ 85351. Approved for 11 hours of category 1 credit toward ArMA, AMA & AAFP CME Certificates.

NOVEMBER

Symposium on Stress
November 3 & 4, 1979. Sheraton-Place Hotel, San Francisco, CA. Sponsor: University of California Schools of Medicine, Nursing, Denistry, and Pharmacy. Contact: Univ. of California, Continuing Education Health Sciences, 24 Kirkham, San Francisco, CA 94143. CME Credit available.

Southern Medical Association Annual Scientific Assembly
November 4-7, 1979. Las Vegas, Nevada. Sponsor: Southern Medical Assoc. & AMA. Contact: AMA Dept. of Meeting Services 535 N. Dearborn St., Chicago, Ill. or Southern Medical Assoc. 2601 Highland Avenue. Biringham, Alabama 35205.

27th Annual Meeting of the Medical Society of the United States and Mexico.
November 14-17, 1979. Doubletree Inn, Tucson, AZ. Sponsor: Medical Society of the United States and Mexico. Contact: Mrs. Carolyn Parsons, Executive Secretry, 3161 N. Pantano Road, Tucson, AZ 85715.

ACLS Provider Course
November 16-17, 1979. Arizona Medical Association offices, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Sponsor: Arizona Affiliate of the American Heart Assoc. Contact: Carol Turner, ArMA Offices, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

Arizona Society of Otolaryngology and Maxillo-Facial Surgery
November 16 & 17, 1979. The Wigwam, Litchfield Park, AZ. Sponsor: Arizona Society of Otolaryngology & Maxillo-Facial Surgery. Contact: Neil O. Ward, M.D., 4550 N. 51st. Ave., Phoenix, AZ 85031.

The Third Annual National Symposium on Aging
November 17 & 18, 1979. Holiday Inn, Golden Gteway, San Francisco, CA. Sponsor: Univ. of California San Francisco. Contact: Univ. of California, Continuing Education Health Sciences, 24 Kirkham, San Francisco, CA 94143.

DECEMBER

8th Annual Seminar: Bone and Joint Diseases
December 8 and 9, 1979, Camelback Inn, Scottsdale, AZ. Sponsor: Maricopa County General Hospital. Contct: Alex Newman, M.D., Program Chairman, Dept. of Radiology, Maricopa County General Hospital, 2601 E. Roosevelt Street, Phoenix, AZ 85008. Approved for 14 hours of category 1 credit.

ACLS Provider Course
December 14 & 15, 1979. ArMA Offies, 810W. Bethany Home Rd., Phoenix, AZ. Sponsor: Arizona Affiliate of the American Heart Assoc. Contact: Carol Turner, ArMA Offices 810 W. Bethany Home Road, Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

1980

FEBRUARY

New Perspectivies in Cerebral Localization
February 7-9, 1980. Hyatt-Regency Hotel, Phoenix, AZ. Sponsor: St. Joseph's Hospital and Medical Center, Barrows Neurological Institute. Contct: Richard A. Thompson, M.D., Chairman, 350 West Thomas Rod, Phoenix, AZ 85013.

Second Annual International Cataract Surgery Symposium
February 18-10, 1980. Sponsor: Arizona Foundation for Ophthalmic Research and Education. Contact: Ronald W. Barnet, M.D., Medical Director, 421 N. 18th St., Ste 105, Phoenix, AZ 85006. (602) 257-1077. Approved for 12 to 22 hours of category 1 credit.

Pediatric Update 1980
February 25-28, 1980. Sponsor: St. Joesph's Hospital and Medical Center, Dept. of Pediatrics. Contact: Ronald A. Christensen, M.D., Dept. of Pediatric Education. St. Joseph's Hospital and Medical Center, 350 W. Thomas Rd., Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

Contemporary Managment of Stroke and Coronary Disese
February 27 - March 1, 1980. Lodges in Taso, new Mexico. Sponosr: Symposia de Santa Fe. Contact: W.J. Levy, M.D., Symposia de Santa Fe, P.O. Box 5175, Santa Fe, New Mexico 85702

MONTHLY OR WEEKLY

Shrine Medics Meeting
Second Tuesday of each month, Doctors Hospital, 5:45 p.m. J. South Classroom. Sponsor: Shrine Medics. Contact: Robert C. Briggs, M.D., 5121 N. Central Ave., Phoenix, AZ 85012.

Office Psychiatry for the Primary Provider
2nd Monday of month, 4811 N. 7th Street, Phoenix, AZ. Sponsor: Arizona Health Plan. Contact: T.R. Bittker, M.D., Box 5000, Phoenix, AZ 85010. Approved for category 1 credit.

Counter Transference Group
Weekly, Thurs. 8—10 p.m. Sponsor: Phoenix Psychiatric Council. Contact: James E. Campbell, M.D., 5051 N. 34th St., Phoenix, AZ 85018. Approved for 2 hours category 1 credit.

Monthly Meeting of Tucson Radiologists
Last Tues. of month. Plaza International, Tucson, AZ. Sponsor: U of A Medical Center, Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., U of A Health Sciences Center, Dept. of, Radiology, Tucson, AZ 85724. Approved for 2 hours category 1 credit.

Pediatric Grand Rounds
Tuesday 7:30-8:30 a.m in Phoenix: 1st Tues.—Phoenix Indian Hospital, 2nd Tues.—Maricopa County Hospital, 3rd Tues.—Good Samaritan Hospital, 4th Tues.—St. Joseph's Hospital. Sponsor: Maricopa County Hospital (Phoenix Hospital's Affiliated Pediatric Program). Contact: J. Kipp Charlton, M.D., 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 1 hour per session category 1 credit.

Review of Forensic Pathology Current Case, Special Topics
Thursday, weekly, 11 a.m., 120 S. 6th Ave., Phoenix, AZ. Sponsor: Arizona Society of Pathologists. Contact: H.H. Karnitschnig, M.D., 120 S. 6th Ave., Phoenix, AZ. Approved for 1 hour per session category 1 credit.

ARIZONA STATE HOSPITAL

2500 E. Van Buren, Phoenix, AZ. 85008. Contact: Arnold L. Kendall, M.D.

A.S.H. Psychiatric Grand Rounds
2nd Wed., 1:00-2:00 P.M., J-6 Conf. Rm., Contact: Dr. Conger & Staff
Clinical-Pathological Conference
3rd Wed., 1:30-2:30 P.M. General Services Bldg., Conf. Rm.
Medical Grand Rounds
4th Wed., 1:00-2:00 P.M., Medical Bldg. Conf. Rm.
Staff Education Conference
Weekly, Wed., 1 p.m., Contact: Howard E. Wulsin, M.D.

BARROW NEUROLOGICAL INSTITUTE

Medical Education
Barrow Neurological Institute of St. Joseph's Hospital and Medical Center, 350 W. Thomas Rd., Phoenix, AZ 85013. Sponsor: St Joseph's Hospital & Medical Center. Contact: John R. Green, M.D. Approved for 1 hour category 1 credit.

Neurology Teaching Conference
Tuesdays, 8:30-9:30 a.m., Eighth Floor Conf. Room.

Neurosurgery Morbidity Conference
Wednesdays, 8:15-9:15 a.m., on first and third weeks, Eighth Floor Conference Room.

Neuro-ophthalmology Conference
Tuesdays, 7:30 a.m. in 8th floor neurology conference room.

Spinal Injury Conference
Wednesdays, 8:15-9:15 a.m., on second and fourth weeks, in Neuropathology Conf. Rm.—a multidisciplinary review of admission by neurosurgeons, orthopedists, and rehabilitation specialists

Neuropathology of Gross Specimens Conference
Thursday, 7:30-8:30 a.m. in the Morgue.

Neurology-Neurosurgical Clinical-Neuroradiological and Neuropathological Conference
Fridays, 8-9 a.m., First Floor Conference Rm.

Neuropathology or Neuroradiology Conferences
Friday, 9 a.m., Neuropathology in Neuropathology Conference Rm., Neuroradiology in First Floor Conference Rm.

Neurorehabilitation Conference
Tuesdays, noon, 8th Floor Conference Rm.

Neurosurgical Journal Club
Saturdays, 9-11 a.m. in Eighth Floor Conference Rm.

CAMELBACK HOSPITAL

5055 N. 34th St., Phoenix, AZ 85018. Sponsor: Camelback Hospital. Contact: Medical Director.

Patient Staffing Conference
Three times weekly.

DESERT SAMARITAN HOSPITAL

1400 South Dobson Road, Mesa, Arizona, Contact: L.A. Rosati, M.D. Approved for category 1 credit.

Clinical Conference - Dept. of Medicine
Weekly Thurs., 12:30-1:30 P.M. Buffet Lunch, Grande Rm.
Medical Education Programs
Wed.,7:00 P.M. Desert Samaritan Hospital, Mesa.
Monthly Visiting Professor Programs
Tuesday 7:00 P.M., Buffett 6:00 P.M.
OB/GYN Medical Staff Conference
Weekly, Monday, 12:30-1:30 P.M. Grande Rm.

Surgical Morbidity & Mortality Conference
2nd Thurs., 7:00 A.M., Grande Rm.

GOOD SAMARITAN HOSPITAL

1033 East McDowell Rd., Phoenix, AZ Contact: Warren A. Hinchcliffe, M.D. Approved for category 1 credit.

Anatomical Path. & Cytology
Weekly, Mondays 4:00-5:00 P.M., S-4 Conf Rm.
Gynecological Section Conference
2nd Monday, 12:30-1:30 P.M., West Rm
Monday Noon Conference
Weekly, 12:00-1:00 P.M. R-2 Conf. Rm.
Mortality/Morbidity, Surgery Comm.
2nd Monday, 11:30-2:00 P.M., West Rm.
Obstetrical Section Conference
1st Monday, 12:30-1:30 P.M., West Rm.
Pulmonary Disese Grand Rounds
Weekly, 12:00-1:00 P.M., D-5 North
Family Practice Conference
Weekly Tuesday, 12:00-1:00 P.M., Fam. Practice Conf. Rm.
Pediatric Grand Rounds
3rd. Tuesday, 7:300-8:30 A.M., West Rm.
Peri-Natal Mortality Conference
3rd Tuesday, West Rm. (May-August).
Surgical Grand Rounds
Weekly, Tuesday 7:00-8:30 A.M. East Rm.
Clinical Cancer Fourm
3rd Wednesday, 12:00-1:00 P.M., West Rm, Contact: John A. Bruner, M.D.
Combined Cardiovascular & Family Practice Conf.
Ist, 2nd, & 4th Wed., 12:00-1:00 P.M., D-5 North.
Dr. Finger's Lecture
Weekly, Wed., 12:30-1:30 P.M., R-2 Conf. Rm.
Learning Disabilities
Weekly, 10:30-12:30 A.M., R-2 Conf. Rm.
OBGYN Dept. Review & Statistics
Weekly, 12:30-3:00 P.M., OBGYN Conf. Rm.
PM&R Grand Rounds
1st. & 3rd. Wed., 12:00-1:00 P.M., R-2 Conf. Rm.
Gynecological Pathology
Weekly Thursday, 1:00-2:00 P.M., S-4 Conf. Rm.
GI Radiology Conference
1st Thursday, 12:00-1:00 P.M., F151, Radiology Conf. Rm.
Neurology Grand Rounds
Weekly Thursday, 9:00-10:00 A.M., D-5 North.
Tumor Treatment Planning Conf.
2nd & 4th Thursdays, 12:00-1:00 P.M., F151 Radiology
Joint Tumor Gyn Conference
2nd Friday, 12:30-1:30 P.M., West Rm.
Kinesology
Weekly Friday, 4:00-5:00 P.M., R-2 Conf Rm.
Medical Grand Rounds
Weekly Friday, 8:00-9:00 A.M., West Rm.
Neuropathy Conference
Last Firday, 1:15-2:15 P.M., S-4 Morgue.
Psychiatry Grand Rounds
Weekly Friday, 11:30-1:00 P.M. , East Rm.

Pediatric Wednesday Seminar
July, Sept., March 12:30-1:30 P.M., East RM.
Pediatric Thursday Seminar
May, June, July, 12:30-1:30 P.M., East Rm.
Phoenix Ultrasound Society Meeting
Sept., thru May,2nd Thursday, 7-9:00 P.M., Med. Ed. Bldg. Contact: Dr. A. Ben-Ora.
PM&R Journal Club
Alt. Fridays, 12:00-1:00 P.M., R-2 Conf. Rm.
Family Practice Grand Rounds
1st. Wed.: April, Aug. & Dec., 7:30-9:00 A.M., West Rm.

KINO COMMUNITY HOSPITAL

2800 E. Ajo Way, Tucson, AZ 85713. Contact: Eric G. Ramsay, M.D. Approved for category 1 credit.

Attending's Hour
5th Monday, 12:30 P.M., Contact: Joseph Goldberger, M.D.
Infectious Disese Conference
3rd Monday, 12:30 P.M., Contact: Larry Lincoln, M.D.
Nephrology Conference
1st Monday, 12:30 P.M., contact: Alan Cohn, M.D.
OB/GYN Dept. Meeting
Weekly Mondays, 7:30 A.M., Contact: Lewis Shenker, M.D.
Psychiatric Conference
2nd & 4th Mondays, Contact: Jose Santiago, M.D.
Surgical Conference
Weekly Mondays, Contact: Ronald Fischer, M.D.
Cardiology Conference
4th Tuesday, 12:30 P.M., Contact: Brendan Phibbs, M.D.
Gynecology Conference
5th Tuesday, 12:30 P.M., Contact Lewis Shenker, M.D.
Neurology Conference
1st & 3rd. Tuesday, 12:30 P.M., Contact: H.W. Buschsbaum, M.D.
Radiology Conference
2nd Tuesday, 12:30 P.M., Contact: Marvin Baker, M.D.
Clinical Pharmacology
4th Wed., 12:30 P.M., Contact: Lee Gardner, Pharm.D.
Clinical Pathology Conf.
2nd Wed., 12:30 P.M., Contact: Philip Altman, M.D.
Gynecology Pathology Conference
Weekly Wed., 7:3'45 A.M., Contact: Lewis Shenker, M.D.
Medical Conference-Mortality & Morbidity
3rd Wed., 12:30 P.M., Contact: Joseph Goldberger, M.D.
Rheumatology Conference
1st Wed. 12;30 P.M., Contact: George Werner, M.D.
Cardiology Conference
1st Thursday, 12:30 P.M., Contact: Brendan Phibbs, M.D.
Clinical Correlation
3rd Thursday, 12:30 P.M., Contact: Elliot Munjack, M.D.
G-I Conference
4th Thursday, Contact: Ray Moldow, M.D.

OB/GYN Sonography/X-ray Conference
Weekly Thursday, 9:15 A.M., Contact: Lewis Shenker, M.D.
Chest Conference
2nd & 4th Fridays, 12:30 P.M., Contact: David Engelsberg, M.D.
Dermatology Conference
3rd Friday, 12:30 P.M., Contact: Peter Lynch, M.D.
Hematology Conference
1st Friday, 12:30 P.M., Contact: Steven Ketchel, M.D.

MARYVALE SAMARITAN HOSPITAL

Continuing Medical Education Program
Every other Wednesday, 7-8 a.m.

MARICOPA COUNTY GENERAL HOSPITAL

2601 E. Roosevelt, Phoenix, Az 85008. Contact: Steven Schaffner, M.D.

Medicine Conference
Daily 12:00-1:00 P.M., Contact: Steven Schaffner, M.D.
Chest Surgery Conference
Weekly Mondays, 1:30 P.M., Satan Cruz Rm.
Obstetrical Problem Conference
Weekly Tuesday, 7:15 A.M., Yavapai Rm
Orthopedic Conference
Weekly Tuesday, 7:30 A.M., Santa Cruz Rm.
Pediatric Grand Rounds
2nd Tuesday, 7:30-8:30 A.M.
Hand Surgery Conference
Weekly Wed., 7:30 A.M., Santa Cruz Rm.
Neurosurgery Discharge Planning
Weekly Wed., 1:30 P.M., Station 42.
OB/Neonatology Seminar
Weekly Wed. 7:15 A.M., Yavapai Room
Psychiatry Conference
Weekly Wed. 11:00-12:00 A.M.
Surgery Conference
Weekly Wed., 7:00-8:00 A.M.
Cardiac/Cardiology Conference
Weekly Thurs. 12:00 P.M., Yavapai Room.
OB/GYN Resident Conference
Weekly Thurs. 12:00 P.M., Yavapai Rm.

GYN, Endocrine Seminar
Weekly Friday, 12:30 P.M., Santa Cruz Rm.
GYN Grand Rounds
Weekly Friday, 1:30 P.M., Santa Cruz Rm.
OB/GYN Surgical Pathology Conf.
Weekly Friday, 7:00 A.M., Yavapai Rm.
Orthopedic X-Ray Conference
Weekly Friday, 7:30 A.M., Santa Cruz Rm.

PHOENIX BAPTIST HOSPITAL & MED. CENTER

6025 N. 20th Ave., Phoenix, AZ 85015. Contact: James L. Grobe, M.D. Approved for category 1 credit.

Medical & Surgical Conference
Weekly Monday, 12;00 Noon
Medical Conference
1st. & 3rd Tuesdays, 12:00 Noon.
Medical & Surgical Conference
Weekly Wed., 12:00 Noon.
Medical & Surgical Conference
Weekly Friday, 12:00 Noon.

PHOENIX INDIAN MEDICAL CENTER

4212 North 16th St. Phoenix, AZ 85016. Contact: Leland L. Fairbanks, M.D. Approved for category I credit.

Pediatric Grand Rounds
1st Tuesday, 7:30-8:30 A.M.
Clinical Staff Teaching conference (Family Practice)
Weekly Wed., 8:00-9:00 A.M., Conf. Rm. A.

PHOENIX MEMORIAL HOSPITAL

1201 S. 7th Ave. Phoenix, AZ. 85036. Contact: George Scharf, M.D., Approved for Category 1 credit.

Monthly Medical Education Seminar
3rd Monday, 6:30 P.M., Kiva Conf. Rm.
Tumor Board Conference
Weekly Friday, 12:00 P.M., Kiva Rm., Contact: Howard Kimball, M.D.

SCOTTSDALE MEMORIAL HOSPITAL

7300 East 4th St., Scottsdale, AZ 85251. Contact: W. S. Williams, M.D. Approved for category 1 credit.

Family Practice Conference
1st Monday, 12:45 P.M.
Morbidity & Mortality Conference
2nd Monday, 12:45 P.M.
Medical Grand Rounds
3rd Monday, 12:45 P.M.
Neurology & Neurosurgery Conference
3rd Monday, 12:30 P.M., Doctors' Lounge
Internal Medicine-Clinical Pathlogical Conference
4th Monday, 12:45 P.M.
Pulmonary Medicine Conference
1st Tuesday, 12:30 P.M., Doctors' Lounge
Cardiology Conference
2nd Tuesday 12:30 P.M., Doctors' Lounge
Surgery Departmental Meeting
3rd Tuesday, 12:15 P.M., Doctors' Lounge
Thoracic & Vascular Surgery Conference
4th Tuesday, 12:30 P.M., Doctors' Lounge
Cardiopulmonary Conference (Didactic)
4th Tuesday, 12:30 P.M., Doctors' Lounge
Peri-Natal Mortality & Morbidity Conf.
2nd Wed., 12:30 P.M., Doctors' Lounge
Trauma Conference
3rd Thurs., 12:30 P.M., Doctors' Lounge
Tumor conference
Last Thursday, 12:30 P.M., Doctors' Lounge

Psychiatry & Behavioral Sciences Conference
1st Friday, 12:30 P.M., Doctors' Lounge
GI Medical-Surgical-Radiological Conference
2nd Friday, 12:30 P.M., Doctors' Lounge
Uronephrology-Oncology
Last Friday, 12:30 P.M.. Doctors' Lounge

ST. JOSEPH'S HOSPITAL PHOENIX

350 West Thomas Road, Phoenix, AZ 85013.
Contact: Joseph C. White, M.D.

OB/GYN Section Conference
3rd & 4th Mondays, 12:30-1:30 P.M., 1st Floor Conf. Rm.
Pediatric Grand Rounds
4th Tuesday, 7:30-8:30 A.M., Contact: J. Kipp Charlton. M.D.

ST MARY'S HOSPITAL & HEALTH CENTER

1601 W. St. Mary's Road, Tucson, AZ 85703.
Contact: see below.

Monthly Specialty Conference-Dept. of Surgery
1st Monday, 7:30 A.M., Century Rm. A. Contact: Med. Staff Office.
Grand Rounds: Medical Surgical, Family Practice, Pathology, Radiology
Weekly Thursday
Cardiology Conference
Weekly Friday, 8:00-9:00 A.M., Century Rm. Contact: Anthony Forte, M.D.

U OF A HEALTH SCIENCES CENTER

Sponsor: U of A College of Medicine, Tucson, AZ 85724. Contact: See below. Approved for category 1 credit.

Anesthesiology Lectures
Weekly Thursday, 4:00-5:00 P.M.
Cardiac Catheterization Conference
Weekly Friday, 4:00 P.M., Contact: Dr. Temkin.
Cardiology Research Conference
Weekly Tuesday, 7:30 A.M., Contact: Dr. Roeske.
Case Discussion
Weekly Wed., 7:00 A.M., Din. Rm.C&D., Contact: Drs. Vaughan & Doyle.
Clinical Immunology, Allergy & Rheumatology Rounds
Every Friday, Noon-1 p.m. Contact: John Boyer, M.D., Dept. of Internal Medicine.
Cerebrovascular Disease Conference
Mondays, 5-6 p.m., weekly, Rm. 5505. Contact: Wesly Moore, M.D.,Depts. of Surgery, Nurology & Radiology.
Dean's Clinical Rounds
Contact: Louis J. Kettel, M.D.

Dermatology Rounds
Weekly Wed., 11:00 A.M., Contact: Dr. Lynch
Ear, Nose & Throat
Alternate Wed., 4:00 P.M., Contact: Dr. Coulthard
Educaton Bloc
Weekly Thurs, 4:00-5:00 P.M., Rm 5403
Endocrinology Seminar
Weekly Thursday, 12:00-1:00 P.M., Contact: Dr. Nugent
Gastrointestinal Path. Conference
4th Friday, 1:30 P.M.
Head & Neck Tumor Meeting
Alternate Wed., 4:00 P.M., Contact: Dr. Manning
Hematology-Oncology Clinical Conference
Every Tuesday, Noon-1 p.m., 1st, 3rd & 5th Tuesdays, Rm 6505, Tucson Medical Center. 2nd & 4th Tuesdays, Rm. N318, VA Hospital. Contact: Sidney Salmon, M.D., Dept. of Internal Medicine.
Joint Thyroid Clinic Conference
1st & 3rd Mondays of every month.1 to 2 p.m., AHSC,Rm. 6505. Contact: Sidney Werner, M.D., Dept. of Internal Medicine, Endocrinology Section.
Nuclear Medicine
Thursday, 7:30 a.m., Radiology Conf. Rm., UAHSC. Contact: Irwin M. Freundlich, M.D., Dept. of Diagnostic Radiology.

Neurology Grand Rounds
Every Friday, Noon, 1st, 3rd, & 5th Fridays, UAHSC, Rm. 7505. 2nd & 4th Fridays, VA Hospital. Contact: William A. Sibley, M.D., Dept. of Neurology.
Neuromuscular Disese Conference
Weekly Friday, 11:00 A.M., Contact: Dr. Stern
Neuropathology Review Conference
Thursdays, 8:30 a.m., Rm. 5120, UAHSC. Contact: Peter C. Johnson, M.D.
Neuroradiology Conference
Weekly Thurs., 5:00 P.M., Contact: Dr. P.C. Christenson
Neurosciences Seminar
Every Friday, 7:30 a.m.,UAHSC, Rm 7505. Contact: Colin R. Bamford, M.D., or Jose F. Laguna, M.D., Dept. of Neurology.
Neuromus Journal Conference
2nd & 4th Thursday, 7:00-9:00 P.M., Contact: Dr. Stern.
Orthopedic Rounds
Saturday, 8:00 A.M., Contact: Dr. Peltier.
Pathology Seminar
Friday, 4:30-5:30 p.m., Rm. 5120, UAHSC. Contact: Paul Finley, M.D., Dept. of Pathology.
Pediatric Problem Patient Conference
Weekly Wed., 8:00 A.M., Contact Dr. Walson
Pediatric Research Forum
Weekly Tuesday, 7:30 A.M., Contact: Dr. James Jones
Pediatric Specialty Conference
Weekly Friday, 8:00 A.M., Contact: Dr. Philip Walson.
Psychiatric Grand Rounds
Weekly Wed., 12:00 Noon, Contact: Dr. Robert Cutts
(Psych.) Monthly Case Conference
2nd Friday, 7:30-9:00 A.M., Contact: Dr. Alan Levens
Pulmonary Rounds
Weekly Friday, 11:30 A.M., Contact Dr. Benjamin Burrowns.
Chest Radiology
Monday, 5-6 p.m., weekly, Rm 1535F, UAHSC. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Gastrointestinal Radiology
Mondays, 7:30 a.m., weekly except for summer months. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Radiation Oncology Planning Conference
Friday, weekly, 8:30-10 a.m., UAHSC, Rm. 0655. Contact: Robert S. Heusinkveld, M.D., Radiation Oncology Section.

Radiology-Rheumatology Conference
Thursday, 8 a.m., UAHSC, Library Rm.1535C. Contact: Irwin M. Freundlich, M.D. Dept. of Radiology.

Renal Pathology Conference
1st & 3rd Thurs., 11:30 A.M., Contact: Dr. Nagle.
Resident's Conference
Wednesday, weekly 5-6 p.m., Diag. Radiology Conf. Rm. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology

Uroradiology
Alternate Thurs, 5:00 P.M., Contact I.M. Freundlich
Surgical Literature/Basic Skills
Saturday, 8:00 A.M., Contact: Dr. P. Whitney
Surgical Morbidity & Mortality Conference
Weekly Wed., 8:00 A.M., Contact: Dr. Wangensteen.
Trauma Conference
Every Thursday,4-5 p.m. UAHSC, Rm. 5505. Contact: Martin Silverstein, M.D., Dept. of Surgery.

Grand Ward Rounds—Trauma
Every Tuesday, 8 a.m., UAHSC. Contact: Martin Silverstein, Surgery Dept., Trauma
Toxicology Conference
Weekly Tuesday, 8:00 A.M., Contact: Dr. Keith Likes
Section.

Monthly Meeting Tucson Ultrasonography Group
First Wed. of month, 4:30-5:30 p.m., UAHSC. Contact: Irwin M. Freundlich, M.D.

General Urology Conference
Weekly Thursday, 12:00 Noon, Contact: Dr. G. W. Drach.

VETERANS ADMINISTRATION HOSPITAL

7th St. and Indian School Rd., Phoenix, AZ 85012. Sponsor: VA Hospital. Contact: Alfred Heilbrumn, M.D. Approved for Category 1 credit.

Cancer Symposium
Weekly Wed., 1:00 P.M., Contact Dr. Berkley, Ext. 419
Cardiology Conference
2nd Thursday — 1 p.m. — T5, Dr. Habibzadeh, Ext. 348.

Clinicopathology Conference
4th Thursday, March, June, Sept. & Dec., 1:00 P.M., Contact Dr. Likos, Ext. 623
Dermatology Conference
1st, 2nd, & 3rd Wednesday (usually, but is subject to change) 10 a.m. — C118, Dr. Roberts, Ext. 426.

PRACTICAL IOL COURSE

Course Director: Samuel L. Pallin, M.D.

For Curriculum Information: Phone (602) 972-1141

Over the last four years, *our course coordinator has arranged over 25 courses in the USA and South America.* The courses this year have featured or will feature, *such well-known surgeons as:*

- Dr. Larry Leiske
- Dr. Marvin Kwitko
- Dr. Malcolm McCannel
- Dr. Samuel L. Pallin
- Dr. Dennis Shepard
- Dr. William M. Bourne
- Dr. C. W. Simcoe
- Dr. Norman Stahl
- Dr. James Gills
- Dr. John M. Graether

These courses are designed to teach the various types of implant surgery. *Video tapes and films will be utilized in teaching the latest techniques and the practical aspects of IOL Surgery.*

The manufacturers of IOL's and several manufacturers of ultrasound equipment, IOL instruments, microscopes, and Phaco emulsifiers *will be invited to display and consult.*

Practical surgery with IOL's, Microscopes and silicone eyes will be utilized!

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(602) 836-7800

☐ **HILTON HEAD, S.C. 29928**
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Palmetto Dunes
P.O. Box 5606
(803) 785-2141

☐ **SANTA BARBARA, CA 93101**
February 16-17, 1980
Mar Monte Hotel & Spa
1121 E. Cabrillo Blvd.
(805) 963-0744

PLEASE KEEP THIS FORM AS A REMINDER

☐ **ALBUQUERQUE, N.M. 87119**
March 21-22, 1980
Airport Marina Hotel
Albuquerque Int'l Airport
(312) 686-8000

☐ **WASHINGTON, D.C. 20036**
April 19-20, 1980
Hilton Hotel
16th and K
(202) 393-1000

☐ **CHICAGO, IL 60666**
May 24-25, 1980
O'Hare Hilton
O'Hare Airport
(312) 686-8000

(805) 965-1085
CALL COLLECT

G.I. Clinic
2nd & 4th Wed, 1:00-4:00 P.M.,

G.I. Radiology Clinical Correlation Conference 1st & 3rd Monday — 1 p.m. — C435, Dr. Sanowski, Ext. 618.

G.I. Grand Rounds
Weekly, Wed., 8:00 A.M., G-I Ward, Contact: Dr. Severino

G.I. Resident Conference
Weekly Friday, 1:00 P.M., D446, Contacts: Drs. Sanowski & Kozarek

G.I. Medical Surgical Conference
Weekly Friday, 2:00 P.M., Path. Conf. Rm.

G.I. Rounds
Weekly Thursday, 1:00 P.M., D446

Medical Service Grand Rounds
Weekly Friday — 11 a.m. — T5, Dr. Zeller, Ext 272

Medical-Surgical G.I. Conference
1st & 3rd Friday, 2:00 P.M., Rm. 2128, Contact: Drs. Kozarek and Mertz, Ext 493

Medical-Surgical Chest Conference
1st and 3rd Thursday — 1 p.m. — T5, Dr. Chowdhury, Ext. 388.

Surgery-Pathology Conference
Weekly Thursday, 2 p.m. — 2128, Drs. Mertz, Ext. 493 & Likos, Ext 623

Surgery Grand Rounds
Weekly Thursday, 3:00 P.M., Contact: Dr. Mertz, Ext. 493.

TUCSON VETERANS ADMINISTRATION HOSPITAL

3601 South Sixty Ave., Tucson, AZ 85723. Contacts listed below. Aprroved for category 1 credit.

Medical Surgical Chest Conference
Weekly Tuesday, 2:00 P.M., Contact: Dr. Young

Medical Grand Rounds
Weekly Wed., 12:00-1:00 P.M., V.A. Hospital Staff Conf. Rm. & (AHSC), Contact: Jay Smith, M.D.

Surgical Grand Rounds
Weekly Wed., 4:00 P.M., Contact: Dr. Putnam

Endocrinology Seminar
1st.,3rd, & 5th Thurdsays, 12:00-1:00 P.M., Rm. N318, Contact: Dept. of Internal Medicine.

Vascular Radiology, Interesting Case Conf.
Weekly Thursday, 12:00 Noon

Carl T. Kirchmaier, M.D.
MEDICAL DIRECTOR

50 YEARS OF SERVICE

In early June, Carl Kirchmaier, M.D., HBA Life's medical director, returned to the University of Cincinnati to attend his graduating class' Fiftieth reunion!

We congratulate "Dr. Carl" on his half-century of dedicated medical service.

HBA Life is fortunate to have a physician of his knowledge and experience on our staff.

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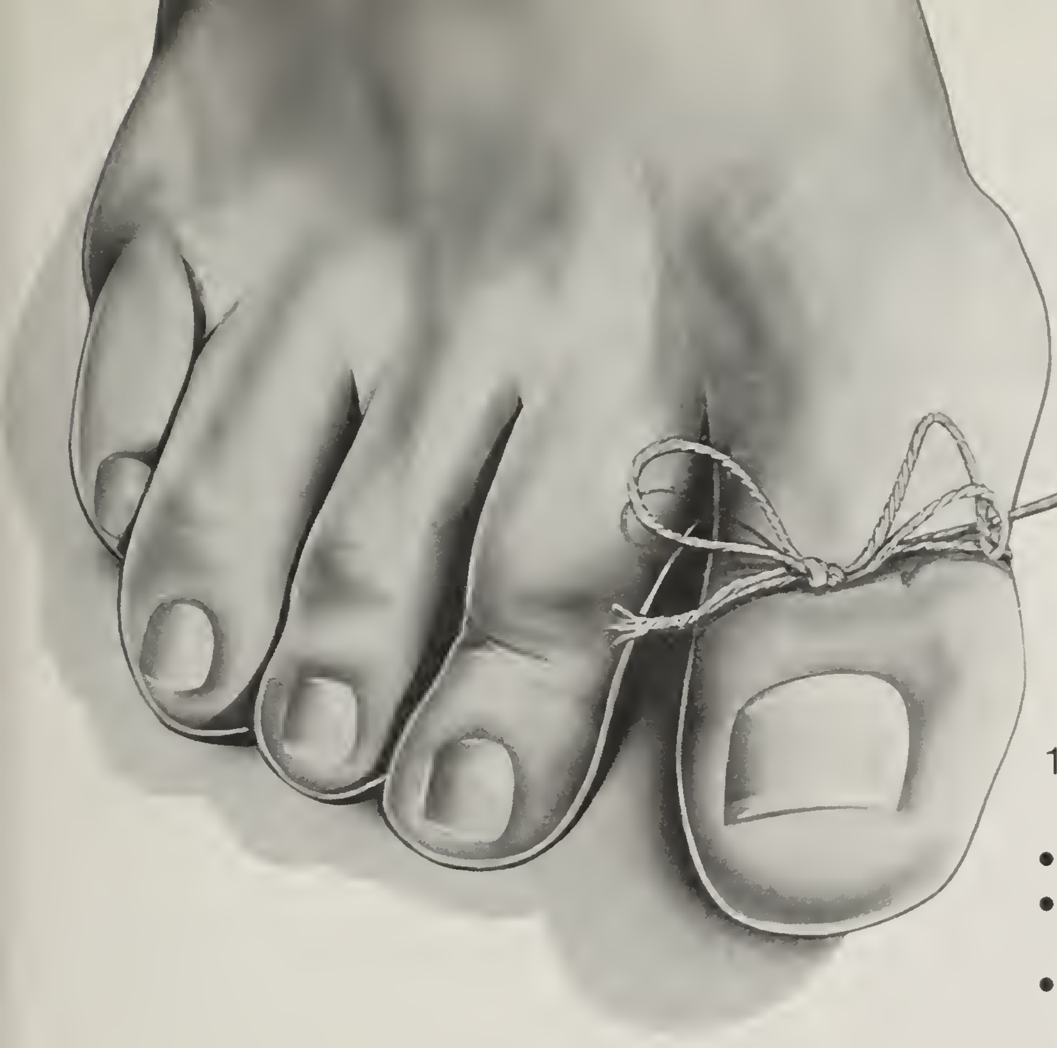

A reminder

ZYLOPRIM®
(allopurinol)

100 and 300 mg scored Tablets

- inhibits uric acid formation
- helps prevent urate crystal depositions in synovia
- reduces risk of uric acid lithiasis

INDICATIONS AND USE: This is not an innocuous drug and strict attention should be given to the indications for its use. Pending further investigation, its use in other hyperuricemic states is not indicated at this time.

Zyloprim® (allopurinol) is intended for:

1. treatment of gout, either primary, or secondary to the hyperuricemia associated with blood dyscrasias and their therapy;
2. treatment of primary or secondary uric acid nephropathy, with or without accompanying symptoms of gout;
3. treatment of patients with recurrent uric acid stone formation;
4. prophylactic treatment to prevent tissue urate deposition, renal calculi, or uric acid nephropathy in patients with leukemias, lymphomas and malignancies who are receiving cancer chemotherapy with its resultant elevating effect on serum uric acid levels.

CONTRAINDICATIONS: Use in children with the exception of those with hyperuricemia secondary to malignancy. The drug should not be employed in nursing mothers.

Patients who have developed a severe reaction to Zyloprim should not be restarted on the drug.

WARNINGS: ZYLOPRIM SHOULD BE DISCONTINUED AT THE FIRST APPEARANCE OF SKIN RASH OR ANY SIGN OF ADVERSE REACTION. In some instances a skin rash may be followed by more severe hypersensitivity reactions such as exfoliative, urticarial and purpuric lesions as well as Stevens-Johnson syndrome (erythema multiforme) and very rarely a generalized vasculitis which may lead to irreversible hepatotoxicity and death.

A few cases of reversible clinical hepatotoxicity have been noted and in some patients asymptomatic rises in serum alkaline phosphatase or serum transaminase have been observed. Accordingly, periodic liver function tests should be performed during the early stages of therapy, particularly in patients with pre-existing liver disease.

Patients should be alerted to the need for due precautions when engaging in activities where alertness is mandatory.

Nevertheless, iron salts should not be given simultaneously with Zyloprim. This drug should not be administered to immediate relatives of patients with idiopathic hemochromatosis.

In patients receiving Purinethol® (mercaptopurine) or Imuran® (azathioprine), the concomitant administration of 300-600 mg of Zyloprim per day will require a reduction in dose to approximately one-third to one-fourth of the usual dose of mercaptopurine or azathioprine. Subsequent adjustment of doses of Purinethol or Imuran should be made on the basis of therapeutic response and any toxic effects.

Usage in Pregnancy and Women of Childbearing Age: Zyloprim® (allopurinol) should be used in pregnant women or women of childbearing age only if the potential benefits to the patient are weighed against the possible risk to the fetus.

PRECAUTIONS: Some investigators have reported an increase in acute attacks of gout during the early stages of allopurinol administration, even when normal or subnormal serum uric acid levels have been attained.

It has been reported that allopurinol prolongs the half-life of the anticoagulant, dicumarol. This interaction should be kept in mind when allopurinol is given to patients already on anticoagulant therapy, and the coagulation time should be reassessed.

A fluid intake sufficient to yield a daily urinary output of at least 2 liters and the maintenance of a neutral or, preferably, slightly alkaline urine are desirable to (1) avoid the theoretic possibility of formation of xanthine calculi under the influence of Zyloprim therapy and (2) help prevent renal precipitation of urates in patients receiving concomitant uricosuric agents.

Patients with impaired renal function require less drug and should be carefully observed during the early stages of Zyloprim administration and the drug withdrawn if increased abnormalities in renal function appear.

In patients with severely impaired renal function, or decreased urate clearance, the half-life of oxipurinol in the plasma is greatly prolonged. Therefore, a dose of 100 mg per day or 300 mg twice a week, or perhaps less, may be sufficient to maintain adequate xanthine oxidase inhibition to reduce serum urate levels. Such patients should be treated with the lowest effective dose, in order to minimize side effects.

Mild reticulocytosis has appeared in some patients.

As with all new agents, periodic determination of liver and kidney function and complete blood counts should be performed especially during the first few months of therapy.

ADVERSE REACTIONS:

Dermatologic: Because in some instances skin rash has been followed by severe hypersensitivity reactions, it is recommended that therapy be discontinued at the first sign of rash or other adverse reaction (see WARNINGS). Skin rash, usually maculopapular, is the adverse reaction most commonly reported.
Exfoliative, urticarial and purpuric lesions, Stevens-Johnson syndrome (erythema multiforme) and toxic epidermal necrolysis have also been reported.
A few cases of alopecia with and without accompanying dermatitis have been reported.
In some patients with a rash, restarting Zyloprim (allopurinol) therapy at lower doses has been accomplished without untoward incident.

Gastrointestinal: Nausea, vomiting, diarrhea, and intermittent abdominal pain have been reported.

Vascular. There have been rare instances of a generalized hypersensitivity vasculitis or necrotizing angiitis which have led to irreversible hepatotoxicity and death.

Hematopoietic: Agranulocytosis, anemia, aplastic anemia, bone marrow depression, leukopenia, pancytopenia and thrombocytopenia have been reported in patients, most of whom received concomitant drugs with potential for causing these reactions. Zyloprim® (allopurinol) has been neither implicated nor excluded as a cause of these reactions.

Neurologic: There have been a few reports of peripheral neuritis occurring while patients were taking Zyloprim. Drowsiness has also been reported in a few patients.

Ophthalmic: There have been a few reports of cataracts found in patients receiving Zyloprim. It is not known if the cataracts predated the Zyloprim therapy. "Toxic" cataracts were reported in one patient who also received an anti-inflammatory agent; again, the time of onset is unknown. In a group of patients followed by Gutman and Yü for up to five years on Zyloprim therapy, no evidence of ophthalmologic effect attributable to Zyloprim was reported.

Drug Idiosyncrasy. Symptoms suggestive of drug idiosyncrasy have been reported in a few patients. This was characterized by fever, chills, leukopenia or leukocytosis, eosinophilia, arthralgias, skin rash, pruritus, nausea and vomiting.

OVERDOSAGE: Massive overdosing, or acute poisoning, by Zyloprim has not been reported.

HOW SUPPLIED: 100 mg (white) scored tablets, bottles of 100 and 1000; 300 mg (peach) scored tablets, bottles of 30, 100 and 500. Unit dose packs for each strength also available.

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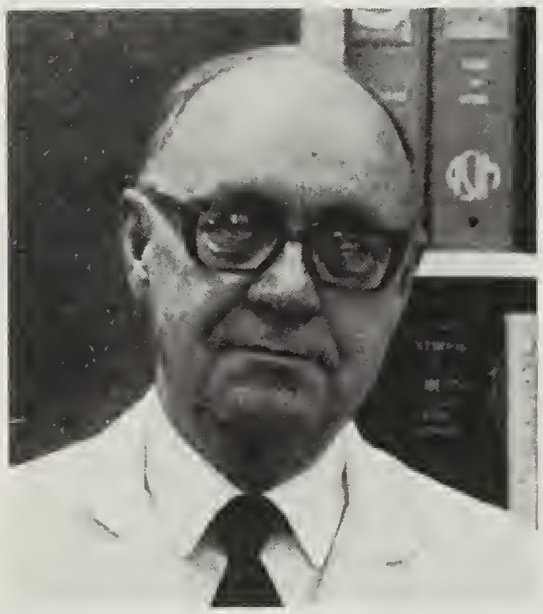

More and more physicians today are beginning to realize the extent of the economic influence they have, and are finding ways of holding costs down.

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What else are physicians doing? Minimizing their patients' hospital stays, whenever possible. Reevaluating routine admissions procedures. Questioning the real need of the diagnostic tests they order for their patients. Avoiding duplicate testing. Trying to discourage their patients' demands for unnecessary medication, treatment or hospitalization. Compiling daily logs of their medical decisions and what they cost. And more.

More physicians today realize what a tough problem we're all faced with. They know this is a challenge for medicine. And that physicians are in the best position to deal with and solve the problem.

**PATIENT CARE Magazine—Outlook 1977 "Face-Off: Cost Containment vs. Chaos," January 1, 1977.*

Lyle CB, et al. "Practice habits in a group of eight internists," ANNALS OF INTERNAL MEDICINE 84 (May 1976), 594-601.

Schroeder SA, et al. "Use of laboratory tests and pharmaceuticals: variation among physicians and effect of cost audit on subsequent use," JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION 225 (Aug. 20, 1973), 969-73.

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The National Cancer Institute will provide the "Helping Smokers Quit" kit free of charge to all physicians who want to participate in this important effort. Included in the kit are guidelines for physicians, a self-test to help smoking patients determine why they smoke, pamphlets with tips on quitting, and waiting room posters to introduce the subject. Each kit contains enough materials to help 50 of your smoking patients who want to quit.

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Adverse Reactions: Drowsiness, ataxia and confusion may occur, especially in the elderly and debilitated. These are reversible in most instances by proper dosage adjustment, but are also occasionally observed at the lower dosage ranges. In a few instances syncope has been reported. Also encountered are isolated instances of skin eruptions, edema, minor menstrual irregularities, nausea and constipation, extrapyramidal symptoms, increased and decreased libido—all infrequent and generally controlled with dosage reduction; changes in EEG patterns (low-voltage fast activity) may appear during and after treatment; blood dyscrasias (including agranulocytosis), jaundice and hepatic dysfunction have been reported occasionally, making periodic blood counts and liver function tests advisable during protracted therapy.

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Capacity will nearly double when the 74-bed Scottsdale Camelback Hospital is completed later this year. This sister facility will offer the same comprehensive psychiatric care... plus serving as the center for an expanded Day Treatment program now concentrated solely at Camelback Hospital Mental Health Center.

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Next door is the Camelback Professional Building where medical practices include general psychiatry, clinical psychology and child/adolescent psychiatry; psychoanalysis; and family counseling. Directory of doctors: George G. Saravia, M.D. • Floyd L. Templeton, M.D. • Stuart M. Gould, Jr., M.D. • Harris D. Murley, M.D. • Paul M. Bindelglas, M.D. • Thomas F. Kruchek, M.D. • Eugene R. Almer, M.D. • Harold E. McNeely, Ph.D. • Otto L. Bendheim, M.D. • W. Walter Fox, M.D. • Stan Goodman, M.D. • Mark A. Wellek, M.D. • James E. Campbell, M.D.

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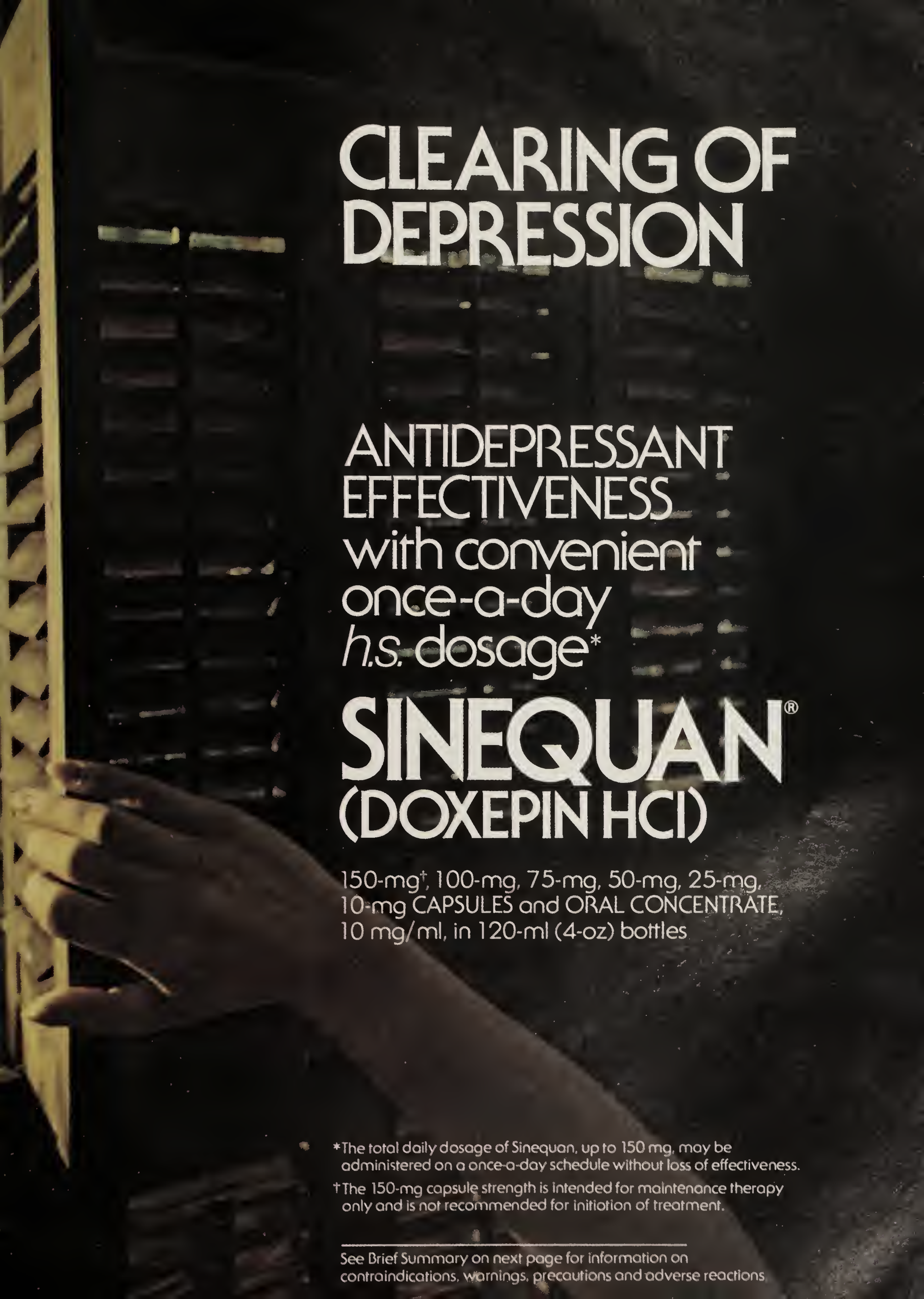

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ANTIDEPRESSANT
EFFECTIVENESS
with convenient
once-a-day
h.s. dosage*

SINEQUAN®
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10-mg CAPSULES and ORAL CONCENTRATE,
10 mg/ml, in 120-ml (4-oz) bottles

*The total daily dosage of Sinequan, up to 150 mg, may be administered on a once-a-day schedule without loss of effectiveness.

†The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

ANTIDEPRESSANT EFFECTIVENESS

SINEQUAN®
(DOXEPIN HCl)

150-mg,* 100-mg, 75-mg, 50-mg, 25-mg, 10-mg CAPSULES and ORAL CONCENTRATE, 10 mg/ml, in 120-ml (4-oz) bottles

CONVENIENT ONCE-A-DAY *h.s.* DOSAGE

which may improve patient compliance. The total daily dosage, up to 150 mg per day, may be given on a once-a-day schedule without loss of effectiveness. Sinequan may also be given on a divided dosage schedule, up to 300 mg per day.

PROMINENT SEDATIVE EFFECT

which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

USUALLY WELL TOLERATED

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have been reported occasionally. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

EXTENDED RANGE OF DOSAGE STRENGTHS

for flexibility in individualizing therapy.

---

* The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients. Possibility of cross sensitivity with other dibenzoxepines should be kept in mind

Warnings. The once-a-day dosage regimen of SINEQUAN (doxepin HCl) in patients with intercurrent illness or patients taking other medications should be carefully adjusted This is especially important in patients receiving other medications with anticholinergic effects

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage This is especially important in patients who may use alcohol excessively

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug

Patients should also be cautioned that their response to alcohol may be potentiated

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy Prescriptions should be written for the smallest feasible amount

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen

Adverse Reactions. NOTE Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use. However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN

Anticholinergic Effects Dry mouth, blurred vision, constipation and urinary retention have been reported If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage

Central Nervous System Effects Drowsiness is the most commonly noticed side effect. This tends to disappear as therapy is continued Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular Cardiovascular effects including hypotension and tachycardia have been reported occasionally.

Allergic Skin rash, edema, photosensitization, and pruritus have occasionally occurred

Hematologic Eosinophilia has been reported in a few patients There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura

Gastrointestinal Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported (See anticholinergic effects.)

Endocrine Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration

Other Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting daily dose of 75 mg is recommended Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response The usual optimum dose range is 75 mg/day to 150 mg/day

In more severely ill patients higher doses may be required with subsequent gradual increase to 300 mg/day if necessary Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg/day

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice Some of these patients have been controlled on doses as low as 25-50 mg/day

The total daily dosage of SINEQUAN (doxepin HCl) may be given on a divided or once-a-day dosage schedule If the once-a-day schedule is employed the maximum recommended dose is 150 mg/day This dose may be given at bedtime. **The 150 mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.**

Antianxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks.

Overdosage.

A Signs and Symptoms

1 Mild: Drowsiness, stupor, blurred vision, excessive dryness of mouth.

2 Severe Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias and tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus), hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes

B. Management and Treatment

1. Mild. Observation and supportive therapy is all that is usually necessary.

2 Severe Medical management of severe SINEQUAN overdosage consists of aggressive supportive therapy If the patient is conscious, gastric lavage, with appropriate precautions to prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbed The use of activated charcoal has been recommended, as has been continuous gastric lavage with saline for 24 hours or more. An adequate airway should be established in comatose patients and assisted ventilation used if necessary EKG monitoring may be required for several days, since relapse after apparent recovery has been reported Arrhythmias should be treated with the appropriate antiarrhythmic agent It has been reported that many of the cardiovascular and CNS symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intravenous administration of 1 mg to 3 mg of physostigmine salicylate Because physostigmine is rapidly metabolized, the dosage should be repeated as required Convulsions may respond to standard anticonvulsant therapy, however, barbiturates may potentiate any respiratory depression Dialysis and forced diuresis generally are not of value in the management of overdosage due to high tissue and protein binding of SINEQUAN

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to: 10 mg, 75 mg, and 100 mg doxepin: bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). 25 mg and 50 mg doxepin bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's) 150 mg doxepin bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN Oral Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibrated at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg Each ml contains doxepin HCl equivalent to 10 mg doxepin Just prior to administration, SINEQUAN Oral Concentrate should be diluted with approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune or pineapple juice SINEQUAN Oral Concentrate is not physically compatible with a number of carbonated beverages For those patients requiring antidepressant therapy who are on methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mixed together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water; but not with grape juice. Preparation and storage of bulk dilutions is not recommended

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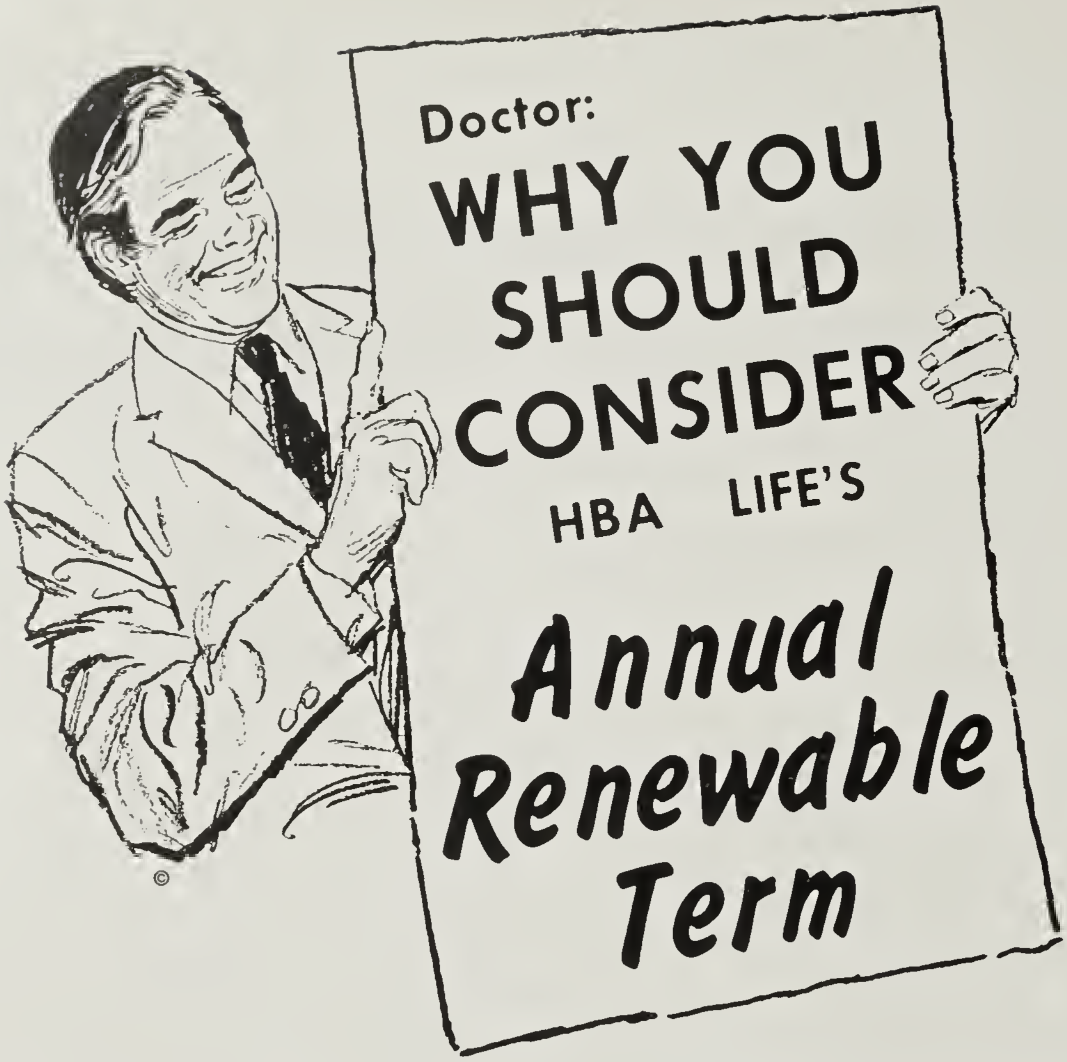

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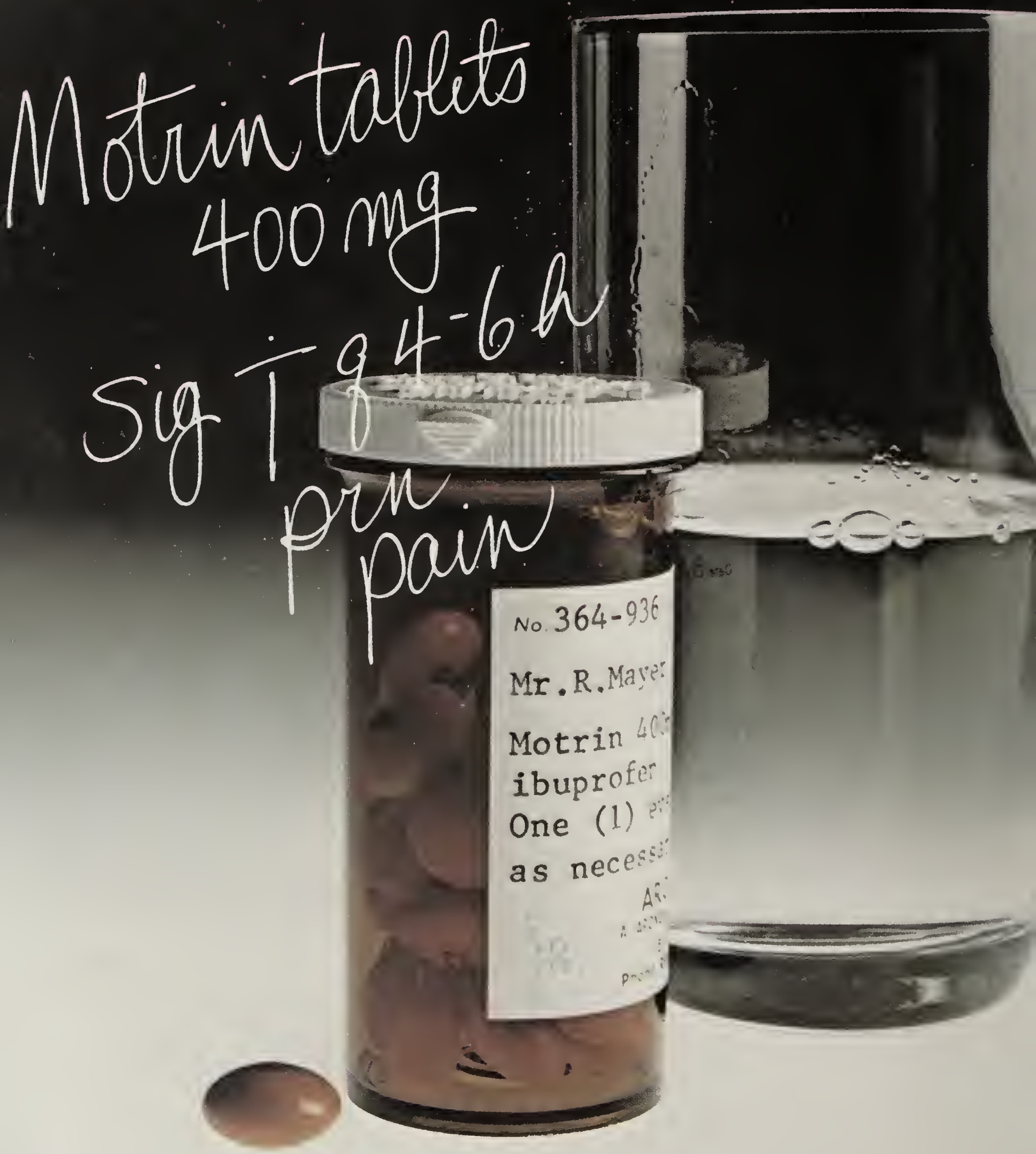
The Upjohn Company
announces
a new
indication for
Motrin®
(ibuprofen)

A well-tolerated, nonnarcotic prescription for pain



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Motrin now proved an effective analgesic for mild to moderate pain

Motrin 400 mg provided greater relief of pain than did propoxyphene 65 mg in controlled clinical pain studies.

Time after drug administration (hour)		.5	1	2	3	4
Mean relief-of-pain scores* (No. patients reporting)	Motrin 400 mg ibuprofen	.89 (108)	1.25 (108)	1.36 (108)	1.28 (107)	1.19 (106)
	Darvon 65 mg propoxyphene	.66 (100)	.99 (99)	1.13 (96)	.99 (96)	.80 (96)
Statistical significance		$p<0.02$	$p<0.01$	$p<0.05$	$p<0.02$	$p<0.002$

*0 = No relief 1 = Partial relief 2 = Complete relief

Data on file at The Upjohn Company

Motrin demonstrated statistically significant greater relief of pain than did Darvon at all time intervals.

Motrin 400 mg TABLETS
ibuprofen, Upjohn

- Not a narcotic • Not addictive • Not habit forming
- Rapid analgesic action • Indicated in acute and chronic pain
- Well tolerated. The most common side effect with Motrin is mild gastrointestinal disturbance.

Please turn the page for a brief summary of prescribing information.

Upjohn

Motrin® (ibuprofen)

now proved an effective analgesic for mild to moderate pain

Motrin® Tablets (ibuprofen, Upjohn)

Indications and Usage: Treatment of signs and symptoms of rheumatoid arthritis and osteoarthritis during acute flares and in long-term management. Safety and efficacy have not been established in Functional Class IV rheumatoid arthritis.

Relief of mild to moderate pain.

Contraindications: Individuals hypersensitive to it, or with the syndrome of nasal polyps, angioedema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory agents (see WARNINGS).

Warnings: Anaphylactoid reactions have occurred in patients with aspirin hypersensitivity (see CONTRAINDICATIONS).

Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Ulceration, perforation, and bleeding may end fatally. An association has not been established. Motrin should be given under close supervision to patients with a history of upper gastrointestinal tract disease, only after consulting ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be tried. If Motrin must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue Motrin and the patient should have an ophthalmologic examination, including central visual fields.

Fluid retention and edema have been associated with Motrin; use with caution in patients with a history of cardiac decompensation

Motrin can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and those on anticoagulant therapy.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain, or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy should have therapy tapered slowly when Motrin is added.

Drug interactions. *Aspirin* used concomitantly may decrease Motrin blood levels. *Coumarin:* Bleeding has been reported in patients taking Motrin and coumarin.

Pregnancy and nursing mothers: Motrin should not be taken during pregnancy or by nursing mothers.

Adverse Reactions

Incidence greater than 1%

Gastrointestinal: The most frequent type of adverse reaction occurring with Motrin is gastrointestinal (4% to 16%). This includes nausea,* epigastric pain,* heartburn,* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of the GI tract (bloating and flatulence). **Central Nervous System:** Dizziness,* headache, nervousness. **Dermatologic:** Rash* (including maculopapular type), pruritus. **Special Senses:** Tinnitus. **Metabolic:** Decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS).

*Incidence 3% to 9%.

Incidence less than 1 in 100

Gastrointestinal: Upper GI ulcer with bleeding and/or perforation, hemorrhage, melena. **Central Nervous System:** Depression, insomnia. **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme. **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure. **Special Senses:** Amblyopia (see PRECAUTIONS). **Hematologic:** Leukopenia, decreased hemoglobin and hematocrit.

Causal relationship unknown

Gastrointestinal: Hepatitis, jaundice, abnormal liver function. **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities. **Dermatologic:** Alopecia, Stevens-Johnson syndrome. **Special Senses:** Conjunctivitis, diplopia, optic neuritis. **Hematologic:** Hemolytic anemia, thrombocytopenia, granulocytopenia, bleeding episodes. **Allergic:** Fever, serum sickness, lupus erythematosus syndrome. **Endocrine:** Gynecomastia, hypoglycemia. **Cardiovascular:** Arrhythmias. **Renal:** Decreased creatinine clearance, polyuria, azotemia.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine, so alkaline diuresis may be beneficial.

Dosage and Administration: Rheumatoid and osteoarthritis, including flares of chronic disease: Suggested dosage is 300, 400 or 600 mg t.i.d. or q.i.d.

Mild to moderate pain: 400 mg every 4 to 6 hours as necessary for relief of pain.

Do not exceed 2400 mg per day.

Caution: Federal law prohibits dispensing without prescription.

For additional product information, see your Upjohn representative or consult the package insert.

MED B-4-S

Upjohn THE UPJOHN COMPANY
Kalamazoo, Michigan 49001 USA

J-7280-4 August, 1979

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ALDORIL®

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and 25 mg HydroDIURIL® (Hydrochlorothiazide, MSD)

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and 15 mg HydroDIURIL® (Hydrochlorothiazide, MSD)

TABLETS
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containing 500 mg ALDOMET® (Methyldopa, MSD)
and 30 mg HydroDIURIL® (Hydrochlorothiazide, MSD)

TABLETS
ALDORIL® D50
containing 500 mg ALDOMET® (Methyldopa, MSD)
and 50 mg HydroDIURIL® (Hydrochlorothiazide, MSD)

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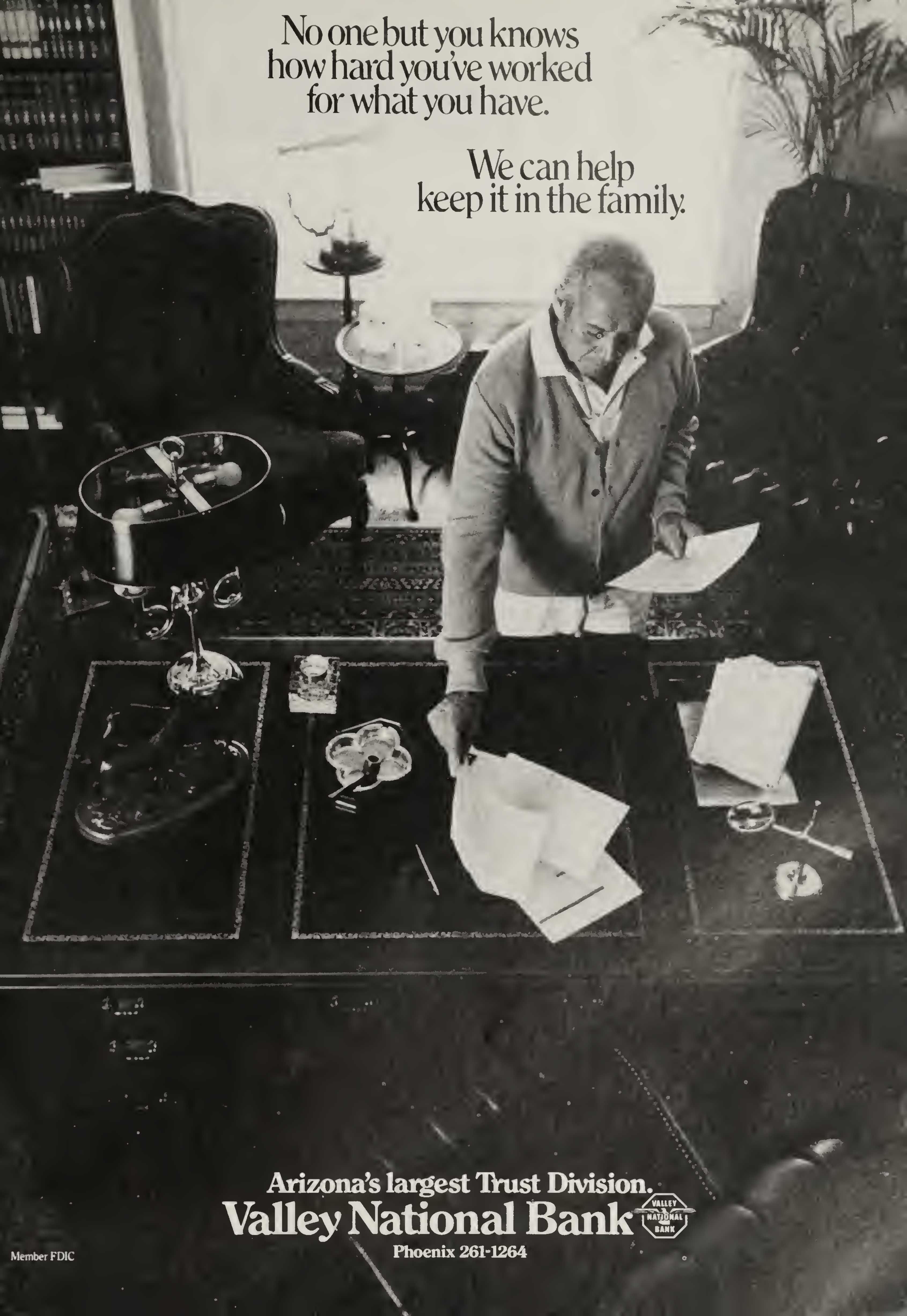
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The Maker

Examining a Few Myths About Prescribing.

Increasing pressure is being put on the practicing physician to prescribe drugs generically. You are told that brand–name products are universally "expensive" and generic versions are relatively "cheap." To make this case, the most extreme (rather than typical) price differentials are cited. Thus, consumers are led to believe that such differentials are commonplace. Even your knowledge and your motives as a physician are questioned.

Understandably, these views have created myths. We think it's time to examine them in the light of all the facts and ramifications.

MYTH: There are no differences in quality and performance between brand-name products and their generic counterparts. The corollary is that there are no differences among products made by high-technology, quality-conscious, research-based companies and those made by commodity-type suppliers.

FACT: The Food and Drug Administration does a good job in monitoring a generally excellent drug supply. Still, it has nowhere near the resources to guarantee the quality and bioavailability of all marketed products at any given time. Just a few months ago, for example, it noted that batches of tetracycline HCl capsules which met official monograph requirements were not bioequivalent to a reference product. As you know, there is substantial literature on this subject affecting many drugs, including such antibiotics as tetracycline and erythromycin. The record on drug recalls and court actions affirms strongly that there are differences among pharmaceutical companies and their products. Research-intensive companies have far better records than those that do no research and may practice minimum quality assurance.

MYTH: Industry favors only "expensive" brand names and denigrates all generics.

FACT: PMA companies make 90 to 95 percent of the drug supply, including, therefore, most of the generics. Drug nomenclature is not the important point; it's the competence of the manufacturer and the integrity of the product that count.

Matters.

TH: Generic options al-
st always exist.

CT: About 55 percent
prescription drug ex-
nditure is for single-
urce drugs. This
eans, of course, that for
ly 45 percent of such
penditure, is a generic
escribing option avail-
le.

TH: Generic
escriptions are filled with
xpensive generics, thus
ving consumers large
ms of money.

CT: Market data show
at you invariably
escribe—and pharma-
sts dispense—both
and and generically
beled products from
own and trusted
urces, in the best inter-
t of patients. In most
ses the patient receives
proven brand product.
vings from voluntary
mandated generic
escribing are grossly
aggerated.

MYTH: Drugs account for a major portion of the rise in health care costs.

FACT: Drugs represent a very small part of such costs. The amount of the health care dollar spent for prescription drugs was about 12 cents in 1967; today it is about 8 cents. And you as a physician are most conscious of how drug therapy can cut hospitalization, avert surgery, reduce office visits and keep patients on the job.

MYTH: Government intrusions into the marketplace will save tax money.

FACT: Government schemes always cost the taxpayer something, and the costs often exceed the benefits. Certainly, any federal "help," such as lists of wholesale drug prices sent to all physicians and pharmacists, will be no exception. Just think of the expense of keeping them current! Moreover, wholesale prices are poor guides to actual transaction prices and even worse guides to retail prices.

The PMA Position

We believe your freedom to prescribe, either by generic or brand name, should be totally unabridged. Otherwise, your prescribing prerogatives and your relationships with patients will be seriously impaired.

The maker does matter

After the myths about price and equivalency have been shattered, one fact stands out more clearly than ever: *The maker does matter.* As always, your best guide to drug therapy for your patients is to select products—both brands and generics—from manufacturers with credentials and performance records you have come to respect.

PMA

Pharmaceutical Manufacturers Association
1155 Fifteenth Street, N.W.
Washington, D.C. 20005

an important
in the treatment of

Efficacy of MONISTAT i.v. therapy...

highly favorable rates of recovery or clinical improvement

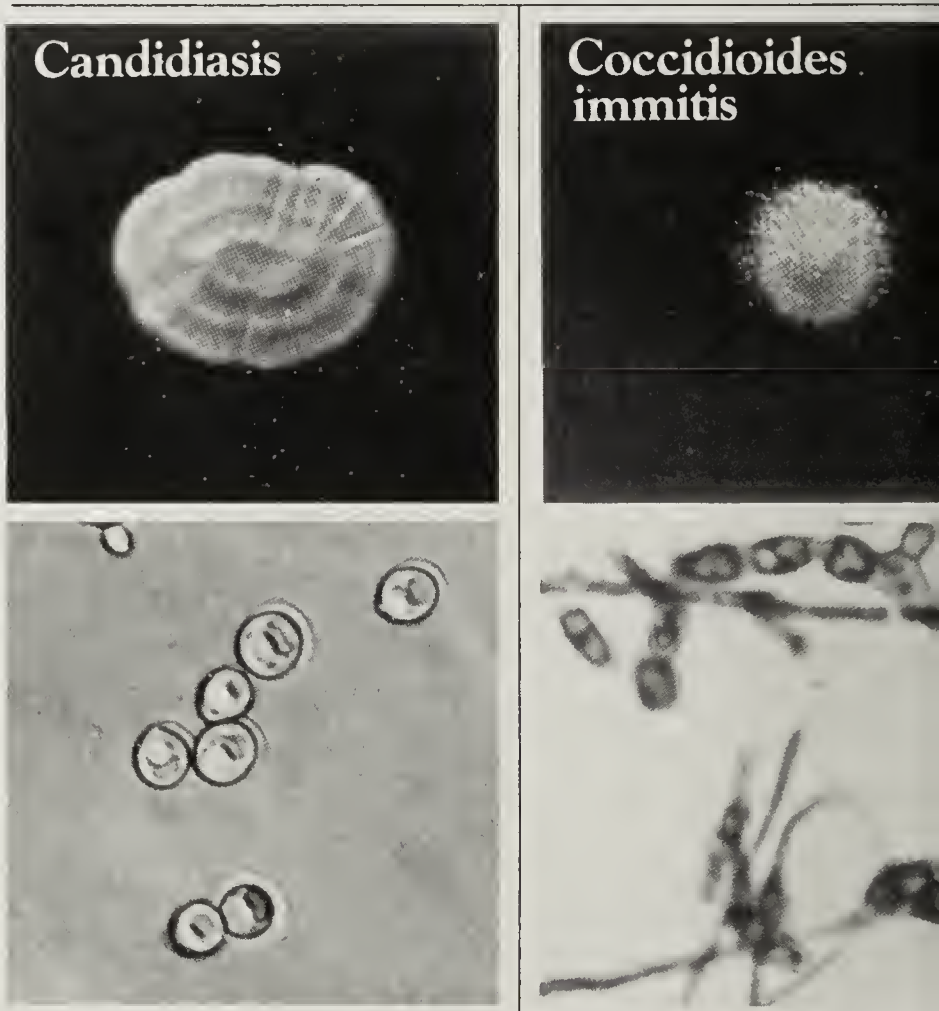
- effective against systemic candidiasis
    chronic mucocutaneous candidiasis
    coccidioidomycosis
    cryptococcosis
    paracoccidioidomycosis
- in patients who did not respond to amphotericin B therapy
- in immunologically-compromised patients with severe infections

rapid onset of clinical improvement

In most patients who responded, improvement was observed within 1-3 weeks of MONISTAT i.v. therapy; however, prolonged treatment courses may be necessary to prevent relapse.

no development of fungal resistance during therapy reported

In vitro resistance of clinical isolates has not been observed during clinical trials.



Photos courtesy of Dubos, J. R., Bacterial and Mycotic Infections of Man, J. B. Lippincott Company, 1968 and Burrows, W., Textbook of Microbiology, W. B. Saunders Company, 19th edition.

and the side effect profile

no nephrotoxicity or other permanent adverse effects in clinical studies

- even in prolonged therapy
- even in patients with renal disease

extensively evaluated

During clinical trials in over 400 patients, no permanent adverse reactions were reported.

Adverse reactions

MONISTAT i.v. should not be administered undiluted. Rapid injection of the undiluted solution may produce transient tachycardia or arrhythmia.

Adverse reactions include phlebitis, pruritus, rash, nausea, vomiting, febrile reactions, drowsiness, diarrhea, anorexia and flushes. Transient hematologic changes have occurred; therefore, the patient should be closely monitored.

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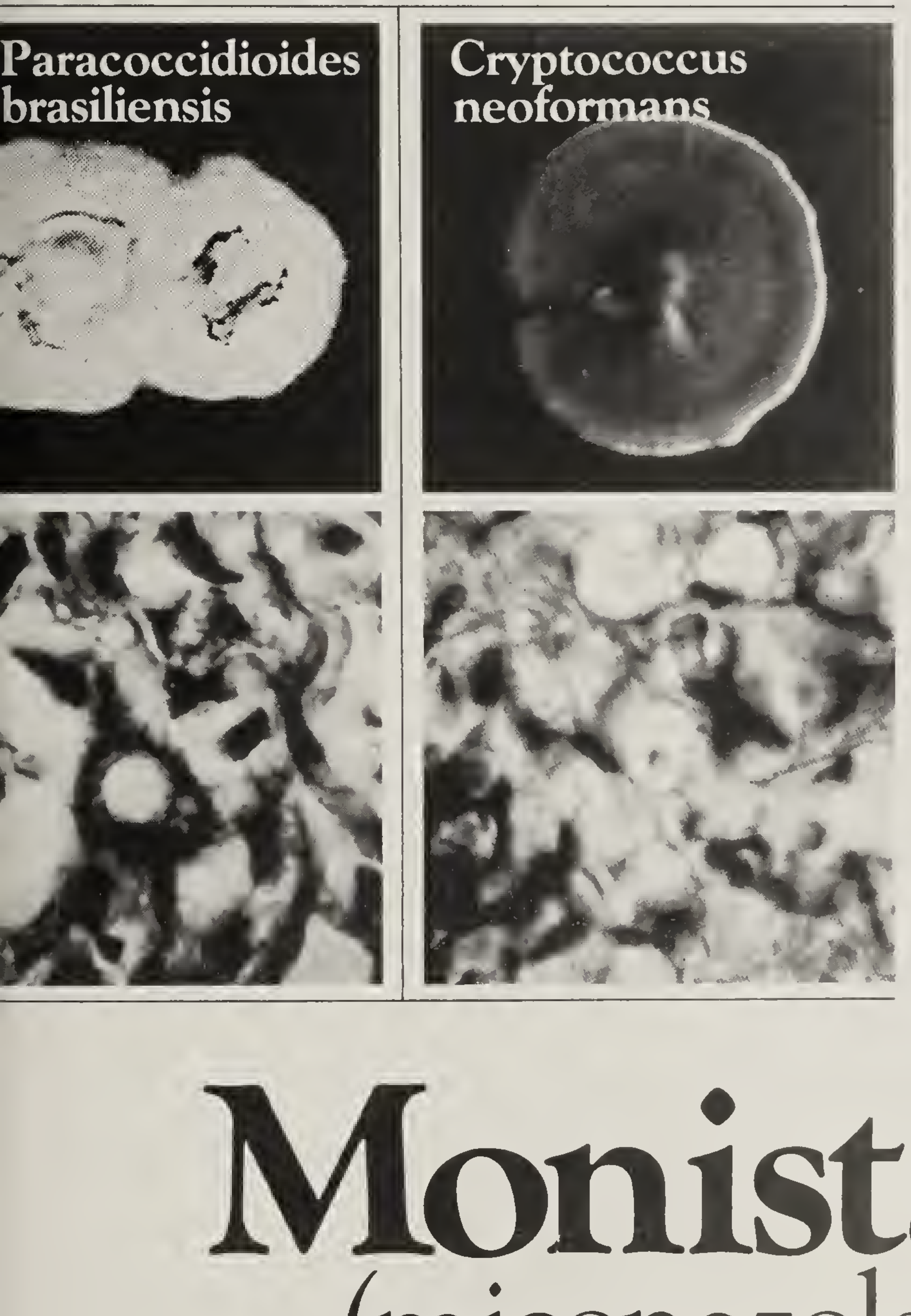
advancement
systemic mycoses



generally well tolerated by severely debilitated patients

- cancer patients
- patients receiving immunosuppressive drugs
- patients with serious underlying conditions such as renal insufficiency or diabetes mellitus

for more information

Because of the uniqueness of MONISTAT i.v. only some of the highlights can be presented in this advertisement. For more information, i.e., product monograph, dosage schedules, etc., please fill out the coupon on the next page and mail to Ortho Pharmaceutical Corporation.

Please turn page for Prescribing Information

Ortho Pharmaceutical Corporation
Raritan, New Jersey 08869

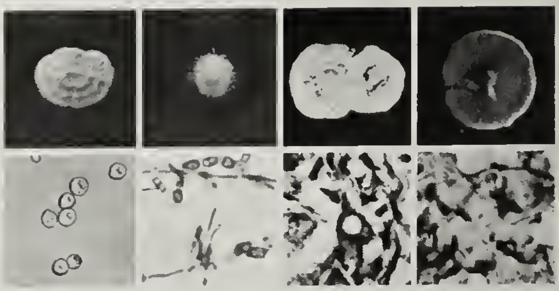
Monistat i.v.

TRADEMARK

(miconazole)

broad-spectrum activity against major systemic fungal pathogens

in systemic fungal infections…



Monistat i.v.

TRADEMARK

(miconazole)

a broad-spectrum systemic antifungal agent

DESCRIPTION: MONISTAT i.v. (miconazole) 1-{2-(2,4-dichlorophenyl)-2-[(2,4-dichlorophenyl) methoxyl ethyl} 1 H-imidazole is a synthetic antifungal supplied as a sterile solution for intravenous infusion. Each ml of this solution contains 10 mg of miconazole with 0.115 ml PEG 40 castor oil, 1.0 mg lactic acid USP, 0.5 mg methylparaben USP, 0.05 mg propylparaben USP in water for injection. Miconazole i.v. is a clear, colorless to slightly yellow solution having a pH of 3.7 to 5.7.

CLINICAL PHARMACOLOGY: MONISTAT i.v. is rapidly metabolized in the liver and about 14% to 22% of the administered dose is excreted in the urine, mainly as inactive metabolites. The pharmacokinetic profile fits a three-compartment open model with the following biologic half-life: 0.4, 2.1, and 24.1 hours for each phase respectively. The pharmacokinetic profile of MONISTAT i.v. is unaltered in patients with renal insufficiency, including those patients on hemodialysis. The in/vitro antifungal activity of MONISTAT i.v. is very broad. Clinical efficacy has been demonstrated in patients with the following species of fungi: *Coccidioides immitis, Candida albicans, Cryptococcus neoformans, and Paracoccidioides brasiliensis.*

Recommended doses of MONISTAT i.v. produce serum concentrations of drug which exceed the in-vitro MIC values for the fungal species noted above. Doses above 9 mg/kg of MONISTAT i.v. produce peak blood levels above 1μg/ml in most cases. The drug penetrates into joints.

INDICATIONS: MONISTAT i.v. is indicated for the treatment of the following severe systemic fungal infections: coccidioidomycosis, candidiasis, cryptococcosis, paracoccidioidomycosis, and for the treatment of chronic mucocutaneous candidiasis. However, in the treatment of fungal meningitis and urinary bladder infections an intravenous infusion alone is inadequate. It must be supplemented with intrathecal administration and bladder irrigation. Appropriate diagnostic procedures should be followed and MICs should be determined.

MONISTAT i.v. should not be used to treat common trivial forms of fungal diseases.

CONTRAINDICATIONS: MONISTAT i.v. is contraindicated in those patients who have shown hypersensitivity to it.

WARNINGS: Rapid injection of undiluted MONISTAT i.v., may produce transient tachycardia or arrhythmia.

PRECAUTIONS: Before a treatment course of MONISTAT i.v. is started, the physician should make sure that the patient is not hypersensitive to the drug product. MONISTAT i.v. should be given by intravenous infusion. The treatment should be started under stringent conditions of hospitalization but subsequently may be given to suitable patients under ambulatory conditions with close clinical monitoring. It is recommended that an initial dose of 200 mg be given with the physician in attendance. It is also recommended that clinical laboratory monitoring including hemoglobin, hematocrit, electrolytes and lipids be performed.

It should be borne in mind that systemic fungal mycoses may be complications of chronic underlying conditions which in themselves may require appropriate measures.

Ortho Pharmaceutical Corporation **AM-10-79**
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Please send me more information on the broad-spectrum, systemic antifungal agent…MONISTAT i.v. (miconazole).

____________________ M.D.

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Specialty____________________

October 1978

Pregnancy: Reproductive studies with MONISTAT i.v. in rats and rabbits revealed no evidence of impaired fertility or harm to the fetus. There are no data, however, on the use of the drug in pregnant women.

Children: Since the safety of miconazole i.v. in children under one year of age has not been extensively studied, its benefits in this age group must be weighed against the possible risks involved.

ADVERSE REACTIONS: Adverse reactions which have been observed with MONISTAT i.v. therapy include phlebitis, pruritus, rash, nausea, vomiting, febrile reactions, drowsiness, diarrhea, anorexia and flushes. In the U.S. studies, 29% of 209 patients studied had phlebitis, 21% pruritus, 18% nausea, 10% fever and chills, 9% rash, and 7% emesis. Transient decreases in hematocrit and serum sodium values have been observed following infusion of MONISTAT i.v. Thrombocytopenia has also been reported. No serious renal or hepatic toxicity has been reported. If pruritus and skin rashes are severe, discontinuation of treatment may be necessary. Nausea and vomiting can be mitigated with antihistaminic or antiemetic drugs given prior to MONISTAT i.v. infusion, or by reducing the dose, slowing the rate of infusion, or avoiding administration at mealtime.

Aggregation of erythrocytes or rouleau formation on blood smears has been reported. Hyperlipemia has occurred in patients and is reported to be due to the vehicle. Cremophor EL (PEG 40 castor oil).

DRUG INTERACTIONS: Drugs containing cremophor-type vehicles are known to cause electrophoretic abnormalities of the lipoprotein. These effects are reversible upon discontinuation of treatment but are usually not an indication that treatment should be discontinued.

Interaction with the coumarin drugs resulting in an enhancement of the anticoagulant effect has also been reported. In cases of simultaneous treatment with MONISTAT i.v. and coumarin drugs, the anticoagulant effect should be carefully titrated since reductions of the anticoagulant doses may be indicated.

DOSAGE AND ADMINISTRATION

DOSAGE

Adults. The doses may vary with the diagnosis and with the infective agent, from 200 to 1200 mg per infusion. The following daily doses, which may be divided over 3 infusions, are recommended.

Organism	Dosage Range*	Duration of Successful Therapy (weeks)
Coccidioidomycosis:	1800 to 3600 mg per day	3 to > 20
Cryptococcosis:	1200 to 2400 mg per day	3 to > 12
Candidiasis:	600 to 1800 mg per day	1 to > 20
Paracoccidioidomycosis:	200 to 1200 mg per day	2 to > 16

*May be divided over 3 infusions.

Repeated courses may be necessitated by relapse or reinfection.

Children: A total daily dose of about 20 to 40 mg/kg is generally adequate. However, a dose of 15 mg/kg body weight per infusion should not be exceeded.

ADMINISTRATION: MONISTAT i.v. should be diluted in at least 200 ml of fluid. The diluent of choice is 0.9% sodium chloride or alternatively Dextrose 5% injectable solution. The intravenous infusion should be given over a period of 30 to 60 minutes.

Generally, treatment should be continued until all clinical and laboratory tests no longer indicate that active fungal infection is present. Inadequate periods of treatment may yield poor response and lead to early recurrence of clinical symptoms. The dosing intervals and sites and the duration of treatment vary from patient to patient and depend on the causative organism.

OTHER MODES OF ADMINISTRATION: Intrathecal. Administration of the undiluted injectable solution of MONISTAT i.v. by the various intrathecal routes (20 mg per dose) is indicated as an adjunct to intravenous treatment in fungal meningitis. Succeeding intrathecal injections may be alternated between lumbar, cervical, and cisternal punctures every 3 to 7 days. Bladder instillation of 200 mg of diluted solution is indicated in the treatment of mycoses of the urinary bladder.

HOW SUPPLIED: MONISTAT i.v. is supplied in 20 ml ampoules.

MONISTAT i.v. (miconazole) is an original product of Janssen Pharmaceutica N.V., Belgium, and co-developed by Ortho Pharmaceutical Corporation.

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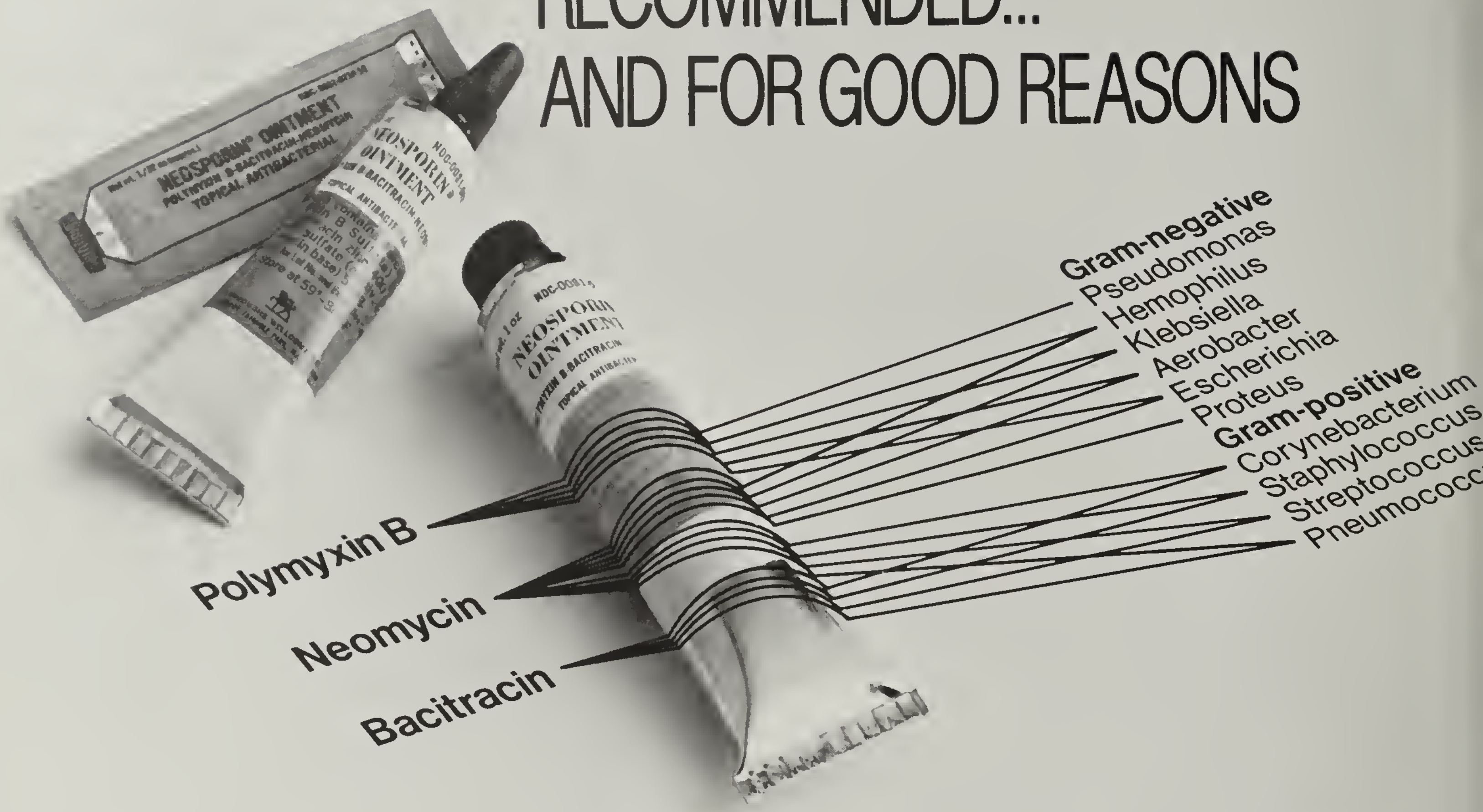

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WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended. When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as a failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

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...in the functional bowel/irritable bowel syndrome*

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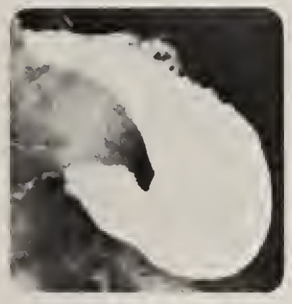
helps control abnormal motor activity with minimal anticholinergic side effects†

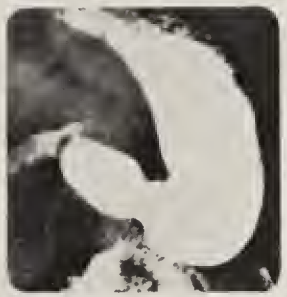
Demonstrated smooth muscle relaxant activity.

In this double-blind study, twenty patients having G.I. series and exhibiting spasm were randomly selected to receive either 2 cc. of Bentyl or sodium chloride intramuscularly. Ten minutes after the injection another radiograph was taken . . .

. . . Bentyl produced definite relaxation in 8 of 10 patients. The sodium chloride produced relaxation in only 3 of 10. No side effects occurred in either group of patients.



Pylorospasm has almost totally blocked passage of barium meal.



Barium meal beginning to pass 10 minutes after intramuscular injection of 20 mg. Bentyl.

"The correlation of spasm relief and drug given was excellent."

*This drug has been classified "probably" effective in treating functional bowel/irritable bowel syndrome

†See Warnings, Precautions and Adverse Reactions.

See following page for prescribing information.

Reference:
King, J.C. and Starkman, N.M.: Evaluation of an antispasmodic. Double-blind evaluation to control gastrointestinal spasms occurring during radiographic examination. A preliminary report. Western Med. 5:356-358, 1964.

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Brief Summary

INDICATIONS

Based on a review of this drug by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the following indications as "probably" effective.

For the treatment of functional bowel/irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis.

THESE FUNCTIONAL DISORDERS ARE OFTEN RELIEVED BY VARYING COMBINATIONS OF SEDATIVE, REASSURANCE, PHYSICIAN INTEREST, AMELIORATION OF ENVIRONMENTAL FACTORS.

For use in the treatment of infant colic (syrup).

Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS: Obstructive uropathy (for example, bladder neck obstruction due to prostatic hypertrophy); obstructive disease of the gastrointestinal tract (as in achalasia, pyloroduodenal stenosis); paralytic ileus, intestinal atony of the elderly or debilitated patient; unstable cardiovascular status in acute hemorrhage, severe ulcerative colitis; toxic megacolon complicating ulcerative colitis; myasthenia gravis WARNINGS: In the presence of a high environmental temperature, heat prostration can occur with drug use (fever and heat stroke due to decreased sweating). Diarrhea may be an early symptom of incomplete intestinal obstruction, especially in patients with ileostomy or colostomy In this instance treatment with this drug would be inappropriate and possibly harmful. Bentyl may produce drowsiness or blurred vision In this event, the patient should be warned not to engage in activities requiring mental alertness such as operating a motor vehicle or other machinery or perform hazardous work while taking this drug. PRECAUTIONS. Although studies have failed to demonstrate adverse effects of dicyclomine hydrochloride in glaucoma or in patients with prostatic hypertrophy, it should be prescribed with caution in patients known to have or suspected of having glaucoma or prostatic hypertrophy Use with caution in patients with: Autonomic neuropathy Hepatic or renal disease. Ulcerative colitis. Large doses may suppress intestinal motility to the point of producing a paralytic ileus and the use of this drug may precipitate or aggravate the serious complication of toxic megacolon. Hyperthyroidism, coronary heart disease, congestive heart failure, cardiac arrhythmias, and hypertension Hiatal hernia associated with reflux esophagitis since anticholinergic drugs may aggravate this condition.

Do not rely on the use of the drug in the presence of complication of biliary tract disease Investigate any tachycardia before giving anticholinergic (atropine-like) drugs since they may increase the heart rate With overdosage, a curare-like action may occur. ADVERSE REACTIONS: Anticholinergics/antispasmodics produce certain effects which may be physiologic or toxic depending upon the individual patient's response. The physician must delineate these. Adverse reactions may include xerostomia; urinary hesitancy and retention, blurred vision and tachycardia; palpitations, mydriasis; cycloplegia; increased ocular tension, loss of taste, headache; nervousness; drowsiness; weakness; dizziness, insomnia; nausea, vomiting, impotence; suppression of lactation; constipation, bloated feeling, severe allergic reaction or drug idiosyncrasies including anaphylaxis; urticaria and other dermal manifestations; some degree of mental confusion and/or excitement, especially in elderly persons; and decreased sweating With the injectable form there may be a temporary sensation of lightheadedness and occasionally local irritation. DOSAGE AND ADMINISTRATION: Dosage must be adjusted to individual patient's needs.

Usual Dosage Bentyl 10 mg. capsule and syrup: *Adults*. 1 or 2 capsules or teaspoonfuls syrup three or four times daily *Children*: 1 capsule or teaspoonful syrup three or four times daily *Infants*: ½ teaspoonful syrup three or four times daily (May be diluted with equal volume of water.) Bentyl 20 mg.: *Adults* 1 tablet three or four times daily Bentyl Injection: *Adults* 2 ml. (20 mg.) every four to six hours intramuscularly only NOT FOR INTRAVENOUS USE. MANAGEMENT OF OVERDOSE: The signs and symptoms of overdose are headache, nausea, vomiting, blurred vision, dilated pupils, hot, dry skin, dizziness, dryness of the mouth, difficulty in swallowing, CNS stimulation. Treatment should consist of gastric lavage, emetics, and activated charcoal Barbiturates may be used either orally or intramuscularly for sedation but they should not be used if Bentyl with Phenobarbital has been ingested If indicated, parenteral cholinergic agents such as Urecholine® (bethanecol chloride USP) should be used.

Product Information as of October, 1978.

Injectable dosage forms manufactured by CONNAUGHT LABORATORIES, INC., Swiftwater, Pennsylvania 18370 or TAYLOR PHARMACAL COMPANY, Decatur, Illinois 62525 for MERRELL-NATIONAL LABORATORIES, Division of Richardson-Merrell Inc., Cincinnati, Ohio 45215, U.S.A.

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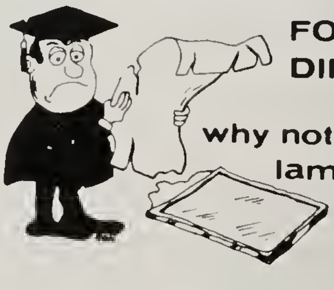

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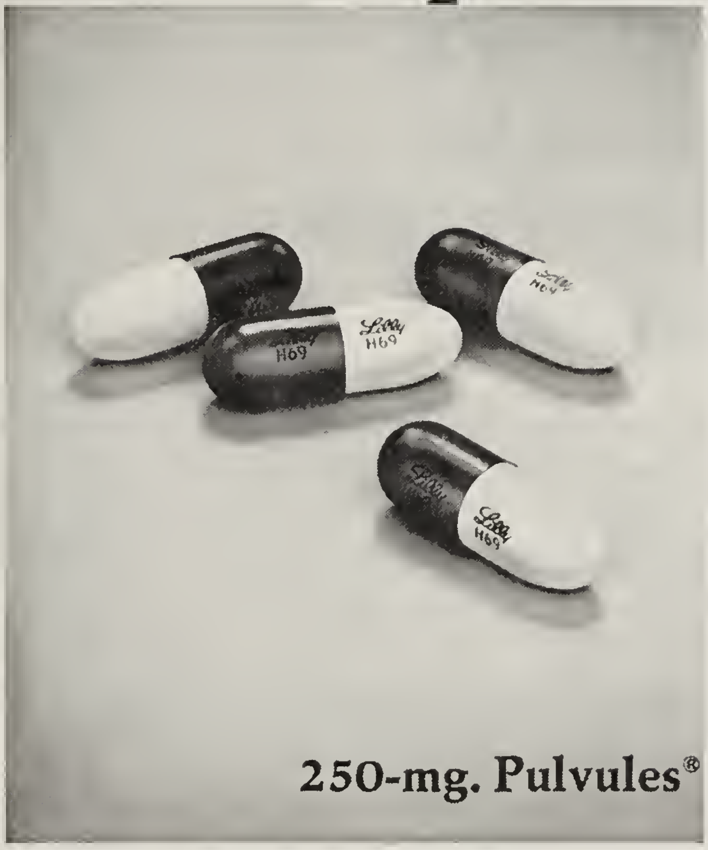

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by Ralph E. Snyder, M.D., F.A.C.P. / Medical Director

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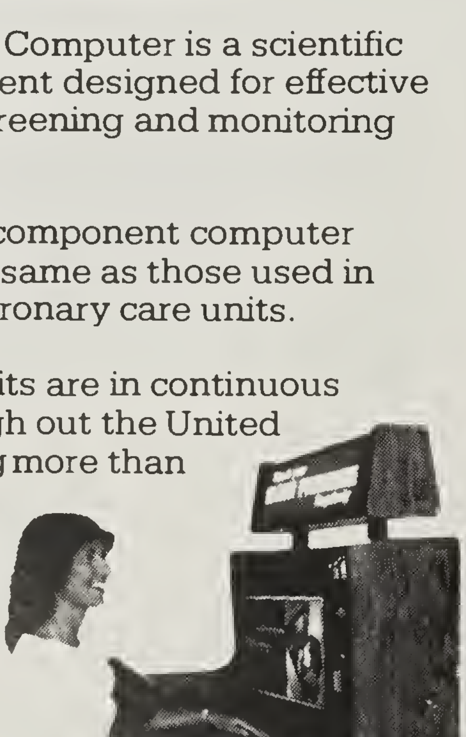

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Joseph H. Chadwick, Ph.D., Director, Health Systems Programs SRI International

"Blood pressure measurements by the Vita-Stat correlated as well as did measurements by physicians and provided reliable determinations suitable for the purposes of screening, referral and monitoring of treatment."

Dr. R. Patterson Russell, Director of the Hypertension Clinic at Johns Hopkins Hospital and Chairman of the High Blood Pressure Commission of Maryland March, 1979 "Preventive Medicine"

***Copies of these and other reports, or medical information about the Vita-Stat Computer is available by writing to me:
Ralph E. Snyder, M.D., Medical Director
2801 75th Street North, St. Petersburg, Florida 33710.

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Cardiology

Increasing Rate of Acute Myocardial Infarction in Southwestern American Indians

Maurice L. Sievers, M.D.
Jeffrey R. Fisher, M.D.

Abstract

Among full-heritage southwestern American Indians, acute myocardial infarction (AMI) was observed significantly more often ($p<0.05$) during 1975-1978 (75/100,000/year, age $>$ 30 years) than in 1957-1966 (35/100,000/year). There is no evidence that this augmentation was the result of improved detection or greater life expectancy. The rise was greater for women than for men, and more marked in the Athapascans (Apache and Navajo) and the Hopi than among the Pimans (Pima and Papago). Contributing factors may be the increasing rates of obesity, diabetes, and hypertension and the rapidly changing lifestyle both on- and off-reservation.

Despite its increase, AMI occurred only about one-fifth as often in these Indians as in the general population of the United States (378/100,000/year). Relative protection from coronary artery disese may be conferred upon southwestern Indians by a lower level of plasma cholesterol and a greater ratio of high-density to low-density lipoprotein cholesterol than in non-Indians. Nonetheless, there is disturbing evidence that AMI is becoming more frequent in these tribes at a time when mortality from coronary artery disease is declining in the general population.

Introduction

Coronary heart disease (CHD) is uncommon in southwestern American Indians[1,2] despite a high prevalence of diabetes mellitus and obesity[3-5] and a moderate but increasing prevalence of hypertension,[3,6] factors augmenting the risk of CHD.[7] This study presents evidence that ischemic heart disease, as manifested by acute myocardial infarction (AMI), has become more frequent in this population since an earlier report by one of the authors for the period 1957-1966.[1] These findings provide an unusual opportunity to examine factors that may be contributing to an escalating rate of AMI in this ethnic group at a time when mortality from CHD in the general U.S. population is declining.[7]

Materials and Methods

The investigation was performed at the 9 Phoenix Area Indian Health Service Hospitals and the one hospital of the Tucson Program Office. It consisted of a prospective study and subsequent review of all medical records and autopsy reports with the diagnosis of AMI, occurring during the four-year period 1975 through 1978. These data were then compared with the incidence of AMI in a study performed during an earlier ten-year period, 1957 through 1966. The earlier investigation of this same population was performed by one of the authors with very similar methodology and diagnostic criteria.[1]

Subjects of the study are from a population of greater than 58,000 southwestern American Indians, of whom an estimated 22,348 were over 30 years of age. The vast majority of these patients receive their medical care from the Indian Health Service. More than 90% have full Indian heritage and, in general, they live on reservations. There are 18 tribes, but almost 80% of the study population are Piman (Pima and Papago), Athapascan (Apache and Navajo), and Hopi.

The criteria for the diagnosis of AMI were the same as in the earlier study[1] — electrocardiographic demonstration of ST-segment elevation with late inversion of T-waves and evolutionary changes on serial tracings, including the appearance of pathologic Q-waves (0.04 seconds or greater in duration). Autopsy evidence of AMI was also accepted. Indians who had coronary thrombosis without infarction, clinical or autopsy evidence of old myocardial infarction, or unexplained sudden deaths were not included. Review of death certificates (for deaths occurring both within hosptials and elsewhere) revealed that unexplained sudden deaths were infrequent (0 to 3 per year).

From: Department of Internal Medicine, Phoenix Indian Medical Center, 4212 N. 16th St., Phoenix, AZ 85016. Address reprint request to Dr. Fisher. The opinions expressed in this paper are those of the authors and do not necessarily reflect the views of the Indian Health Service.

Although some nonsouthwestern Indians and others of predominantly non-Indian ancestry had AMI during this study period, they were excluded from the study.

Standards for the diagnosis of diabetes mellitus are described in a previous publication.[5] Hypertension was considered present when the blood pressure exceeded 150 mm Hg systolic or 90 mm Hg diastolic on several determinations. Total and lipoprotein cholesterol levels were measured on fasting plasma specimens from 20 consecutive Indians, at least 3 months after their myocardial infarcts. Details of the methods of testing and statistical analysis are included in a publication of Howard,[8] in whose laboratory the tests were performed.

The Chi-square test was used for statistical analysis of the AMI results, and beyond the 95% confidence level ($p<0.05$) was considered significant.

Results

During the four-year period of 1975 through 1978, AMI was discovered in 67 southwestern Indians, as compared with 56 during the earlier ten-year (1957-1966) period. In the present study, the mean annual number (16.8) of infarcts was three times the previous yearly average (5.6). However, during the interval between observations the population at risk also increased. As a result, the mean annual rate of infarction for southwestern Indians 30 years of age or older was 75/100,000 for the recent period and 35/100,000 for the earlier investigation, a two-fold increase ($p<0.05$) (Figure 1).

In the present study there were 40 men (60%) and 27 women (40%), as compared with 42 men (75%) and 14 women (25%) in the earlier investigation. The age range in the present study was 40 to 90 years (means: men, 61.4 years; women, 64.1 years). The overall mean age (62.5 years) remained unchanged for the two observation periods. More than half (21/40, 53%) of the men and almost one-third (8/27, 30%) of the women with AMI were over 70 years of age, while only 7% of the affected Indians were 45 years or less. Thus, among southwestern Indians, myocardial infarction continued to afflict predominantly the elderly. The population aged 30 years and older increased 41% between observation periods, while hospital admissions for this age group increased 25%. However, the average annual number of AMIs rose a disproportionate 200%.

The rate of AMI for the Indian women more than tripled between periods (Figure 1). Indeed, the recent annual rate for women (59/100,00) somewhat exceeds the earlier rate for men (52/100,000). During the past 20 years the proportion of women in the southwestern Indian population over age 30 years has increased from 48% to 51%, but this change does not account for the significant rise in the number of myocardial infarcts in the women ($p<0.05$).

Tribal distribution of AMIs is shown in Figure 2. The population distribution for each of the three tribal groupings is similar (Piman 34.9%; Athapascan 31.5%; and, other southwestern tribes 33.6%). Compared with the previous study, the recent infarction rate for Pimans (Pimas and Papagos) remains higher than for the Athapascans (Apache and Navajo). Nevertheless, much greater increases have occurred, in the recent period, among the Athapascans (193%) and other southwestern tribes (194%) than in the Pimans (48%). The "other southwestern" group is composed of largely unrelated tribes. All have small populations except for the Hopi. This tribe had 19% of the population in the "other southwestern" category, but experienced 35% (10/29) of the infarctions for that group. No significant differences in mean age, sex distribution, or degree of Indian heritage were present among the various tribes. Furthermore, the proportion of hospital admissions by tribe did not vary materially between the two study periods.

The mean annual rate of AMI was more than 5 times higher for the diabetic (168/100,000) than for the nondiabetic (31/100,000) southwestern Indians

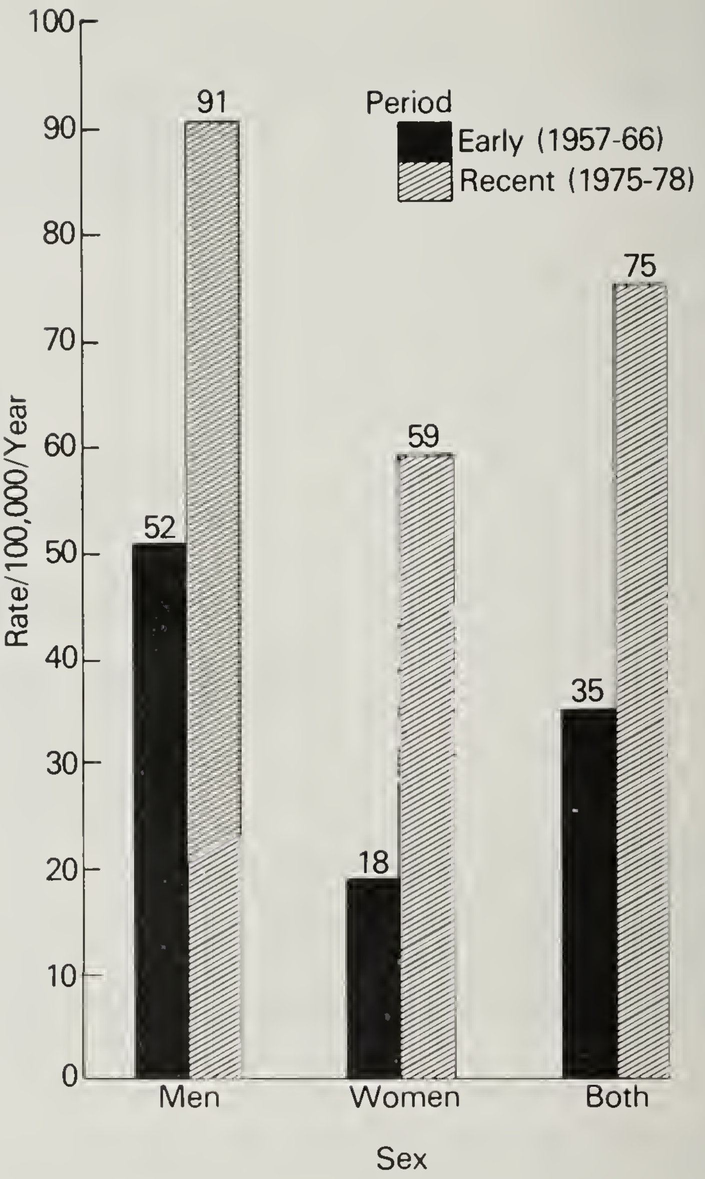

Figure 1
Comparative rates, by sex, for acute myocardial infarction in southwestern American Indians (age ≧ 30 years) during early (1957-66) and recent (1975-78) study periods.

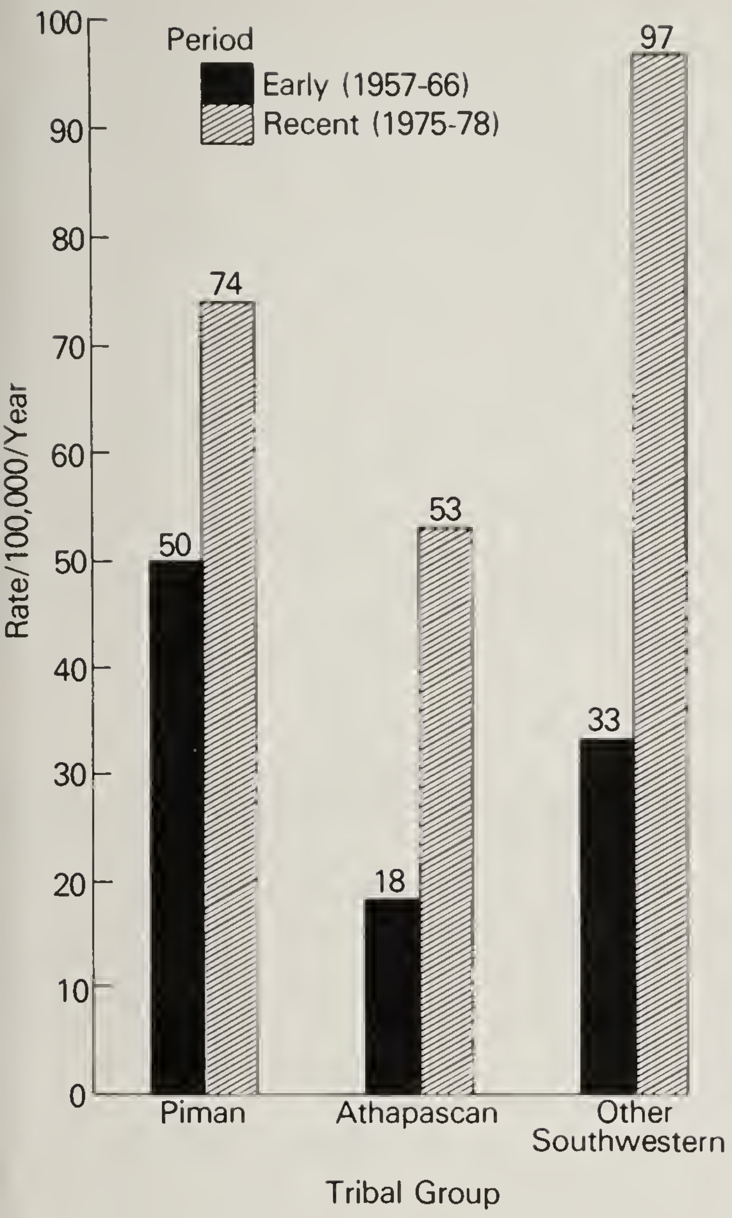

Figure 2
Comparative rates, by tribe (age ≧ 30 years), for acute myocardial infarction during early (1957-66) and recent (1975-78) study periods.

($p<0.01$). This ratio was greater for women (7.0) than for the men (4.9). Despite the inherent bias toward an increasing prevalence of diabetes and hypertension among hospitalized persons, the rates of both of these conditions, for every tribe, were much lower among age- and sex-matched hospitalized medical patients without infarction than among the study subjects. The proportion of persons with AMI who had diabetes mellitus increased between observation periods from 66% to 70%, and the prevalence of hypertension, from 40% to 54%.

In the recent study period, diabetes, hypertension, or both conditions were present in the women (93%) much more often than in the men (75%). Furthermore, of the 12 patients without diabetes or hypertension, 7 were over 60 years of age. Thus, one or more of three risk factors (diabetes, hypertension, or old age) were present in 93% of southwestern Indians with AMI (35/40, 88% of men; 27/27, 100% of women). Lipid studies, done in 20 consecutive patients at least 3 months after their infarcts, revealed that the 8 women, as compared with the 12 men, had both a higher ($p<0.05$) cholesterol (227 ± 18 mg/dL versus 192 ± 14 mg/dL; mean ± SEM) and a lower ($p<0.01$) ratio of high-density (HDL) to low-density lipoprotein (LDL) cholesterol (0.30 ± 0.03 versus 0.41 ± 0.03).

Discussion

It is unlikely that the recently observed increased rate of AMI in southwestern Indians is an artifact related to improved detection.Indeed, gastric carcinoma and pernicious anemia, two other diseases of the older age group, have shown no significant change in frequency between the two observation periods.[3,9] Nor is it likely that large numbers of southwestern Indians with AMI have failed to seek medical care; sudden unexplained death (as determined from review of death certificates) was infrequent (0 to 3 per year) in both the early and recent periods. Furthermore, the rate of autopsies performed during the earlier study (44% of hospital deaths) was more than twice as great as in the present investigation (20%). This decrease in autopsies could bias the data toward discovering acute infarcts less often in the recent period, whereas, in fact, they were found more frequently.

Despite an increasing incidence, myocardial infarction occurs at a much lower rate in southwestern American Indians than among the general population of the United States. In a 1974 survey of discharages from short-stay non-Federal hospitals, conducted by the National Center for Health Statistics,[10] AMI was found in 378/100,000/year among the population aged 30 years or older, in contrast to the 75/100,000/year for Indians in the present study, and 35/100,000/year in the earlier report.

There are several possible reasons for the infrequency of CHD in southwestern Indians. These tribes have significantly lower plasma cholesterol than age- and weight-matched Caucasians, and the levels rise very little with advancing age.[11, 12] The high rate of hepatic cholesterol secretion and the associated increased bile lithogenicity found in certain tribes suggest that their low plasma cholesterol levels may be related to augmented cholesterol disposal. There is also evidence for a decreased synthetic rate of LDL and a higher ratio of HDL to LDL cholesterol in Indians than in Caucasian controls.[13] Furthermore, interpersonal competitiveness has been relatively infrequent among southwestern tribes, who also seldom have had duodenal ulcer disease.[3,9] Southwestern Indians rarely smoke cigarettes, and few do so extensively.[14] In addition, it has been suggested that genetic factors associated with blood group O, the most prevalent group among southwestern Indians (80%), may be associated with a decreased risk of CHD.[15]

Although the rate of myocardial infarction among southwestern Indians is low, as confirmed by autopsy findings,[1, 2] data from both the present and previous studies probably somewhat underestimate the frequency. Adherence to strict electrocardiographic criteria may lead to under-diagnosis by as much as 40%.[16] In addition, diabetes mellitus is extremely prevalent in most tribes, and is associated with an increased rate of silent infarction.[17] Such silent events would not be recorded in this study.

The observed frequency of hypertension, diabetes, and obesity have increased between study periods for many tribes.[4-6] In general, the extent of tribal increases in rates of AMI parallels the rise in rates of diabetes and hypertension, this augmentation being less for the Pimans and greater for the Athapascans and other southwestern tribes. The diet consumed by southwestern American Indians does not differ substantially from that of the general population of the United States, except for more fiber and a relative excess of calories.[3, 18]

Especially noteworthy is the greatly increased rate of myocardial infarction for the Indian women, which has more than tripled between periods (Figure 1), although remaining substantially below the rate for Indian men and for either sex of the general population. Data from the Framingham study[19] indicate that diabetes mellitus has a greater impact on CHD in women than in men. Findings of the present study also show that diabetes augments the rate of AMI to a greater extent in Indian women than in the men. Myocardial infarction was 7 times more frequent for the diabetic women and 4.9 times more frequent for the diabetic men than for the nondiabetic Indians of each sex, respectively. In the present investigation 78% of the Indian women with infarcts had diabetes. All except one of the women were post-menopausal, but none of them was receiving estrogen therapy at the time of infarction. The Indian women with AMI had higher plasma cholesterol levels and a lower ratio of HDL to LDL cholesterol than the men with infarcts. Furthermore, studies of Pima Indians indicate that the diabetic women have lower levels of HDL cholesterol and greater levels of LDL cholesterol than the nondiabetic women.[8] Thus, the relatively high proportion of Indian women with AMI may, in part, be explained by their great prevalence of diabetes and the associated lipoprotein abnormalities.

The increasing frequency of AMI in southwestern Indians has accompanied a vastly changing lifestyle. Differences between living conditions on- and off-reservation have rapidly narrowed. For example, in 1959 at least 60% of the White Mountain Apaches lived in wickiups, the traditional tribal dwellings.[20] In recent years, such housing has become a rarity. Modernization has occurred with apparent decreased physical activity, substantial cultural disruption, and increased psychological stress. Indeed, adaptation to abrupt social changes may be related to the occasional recent occurrence of duodenal ulcer, which was previously rare.[3,9] The increased incidence of both doudenal ulcer and acute myocardial infarction has been more marked for the Athapascans, who were traditionally nomadic hunters, than for the less aggressive agrarian Pimans. Tribes with the greatest rise in infarction rates between the two observation periods also had the largest increases in the frequency of obesity, diabetes mellitus, and hypertension. Southwestern Indians have relatively low levels of coronary artery disease, but despite their decreased vulnerability, they have the same predisposing risk factors as other ethnic groups.

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Seminars in
Chest Medicine

Bronchopulmonary Dysplasia

Chronic Lung Disease Following Neonatal Respiratory Disorders

Bruce G. Nickerson, M.D.
Lynn M. Taussig, M.D.

Editors:
Faysal M. Hasan, M.D.
Richard I. Lemen, M.D.
Bernard E. Levine, M.D.

Bronchopulmonary dysplasia (BPD) is a relatively new and common disease of infancy with numerous medical, social and economic consequences. BPD is the chronic lung disease that sometimes afflicts premature infants who survive hyaline membrane disease (or other neonatal respiratory disorders) and its treatment. It is a new disease because such infants did not survive before the recent advances in neonatology five to ten years ago. This article will review the pathophysiology of this disease, its complications and its treatment. Because it is a new disease there are still many unanswered questions.

BPD is typically found in infants who were very premature (less than 32 weeks gestational age), very small (less than 1500 gm), and very sick with hyaline membrane disease. Incidence figures vary with the criteria used to define BPD, the population studied and the practices of the various intensive care nurseries, but are on the order of 2 to 40 percent of permature infants who require mechanical ventilation.[1-3] The advent of constant positive airway pressure (CPAP) and the acquisiton of greater experience with ventilator managment seems to have decreased the incidence.[2,4]

Table 1
Contributing Factors to Development of BPD

Most Likely	Possible
Degree of prematurity	Fluid Load
Individual susceptibility	Drugs
Inspired oxygen levels	Patent ductus arteriosus
Airway pressure	Infection
Duration of exposure to oxygen and high airway pressures	Intubation
	Interstital emphysema
	Vitamin E deficiency
	Low levels of enzymes
	Sex (males > females)
	Genetic factors

The etiology of BPD is still controversial although it is probably a combination of factors (Table 1). High percentages of inspired oxygen are toxic to the alveoli and pulmonary capillaries of adults and animals.[5-7] Most, but not all, infants with BPD have received greater than 50 percent inspired oxygen in the first few days of life. The trauma of intubation and the barotrauma of mechanical ventilation with high inspiratory pressures also contribute to airway and parenchymal damage.[8-11] The degree of prematurity is important not only because it is correlated with the severity of RDS and hence the need for high inspired oxygen concentrations and mechanical ventilation, but also because the poorly developed surfactant mechanism lead to airway and alveolar collapse. The early onset of continually active breathing in prematures may by itself disrupt the development of alveoli which begin to appear only at 26 weeks gestation.[12] The tiny airways of a 1000 gm infant may be blocked by amounts of mucus or edema that would be less significant to a full-term infant. The roles of infection, vitamin deficiency, low levels of certain enzymes, patent ductus arteriosus, ischemia, fluid overlead, drugs and genetic factors as contributing to the development of BPD have been the subject of speculation and are currently being investigated.[13-16]

BPD was originally divided by Northway[17] into four stages according to radiographic findings. Since then the pathologic, clinical, and physiologic correlates of these stages have been elucidated.[18-20]

Stage one consists of the first two to three days of life and constitutes of classic respiratory distress syndrome. The baby requires oxygen and assisted ventilation; hypoxia is more of a problem than CO_2 retention. The chest radiograph reveals microatelectasis giving a "ground glass" appearance with air bronchograms and poor expansion. Pathologically there are hyaline

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From: Pediatric Pulmonary Section, Dept. of Pediatrics and Division of Respiratory Sciences, Univ. of Arizona Health Sciences Center, Tucson, AZ 85724.

Table 2
Spectrum of BPD
Severity of BPD

	Minimal	Mild	Moderate	Severe
Chest radiograph	Normal	Normal	Hyperexpansion	Hyperexpansion; fibrosis; cysts
Pulmonary function tests	Normal	Slow gas mixing	Slow gas mixing ↑ lung volumes	Slow gas mixing ↑ lung volumes
Clinical aspects	Off oxygen by 30 days	Low concentrations of O_2 required after 30 days	Prolonged O_2 requirements. Mild CC_2 retention	Marked CO_2 retentio Prolonged ventilator a O requirement
Increased risk of developing severe LRI* often requiring hospitalization	Perhaps	Yes	Yes	Yes (Usually with resp tory failure)

* *LRI - lower respiratory infection*

membranes lining the alveoli, atelectasis, loss of ciliated cells in the airways and necrosis of the bronchiolar epithelium.

During stage two (from days four to 10) there is a plateau of the respiratory symptoms instead of the usual resolution of the respiratory distress syndrome. Inspired oxygen requirements decrease but still may be greater than 40%. The infant will produce greater amounts of sputum which may prevent or complicate extubation. The chest radiograph will show a radiodense "whiteout." Pathologically there are persistent hyaline membranes with necrosis and repair of the alveolar epithelim. Much of the mucosa of the small airways desquamates which may contribute to the sputum produced. The findings are similar to those of a "bronchiolitis obliterans." Capillary leakage leads to interstitial pulmonary edema.

During stage 3 BPD (from days 10 to 30) the infant may improve somewhat but often will retain CO_2 even as the oxygen requirements decrease. Fluid retention often becomes a problem and may cause rapid fluctuations in the infant's condition. The chest radiograph shows small cystic lesions alternating with areas of consolidation. The diaphragms that were elevated during the RDS stage begin to flatten as the lungs become hyperexpanded. Pathologically, the hyaline membranes are disappearing, the alveoli are beginning to regenerate but the airway epithelia undergoes squamous metaplasia with flat or "heaped-up" cells replacing the usual columnar epithelium. Bronchial mucus glands are now hyperplastic. There are areas of emphysema alternating with atelectasis and interstitial edema is usually still evident. Some infants may recover during this phase while the more severely involved progress to stage 4.

Carbon dioxide and fluid retention with intermittent wheezing are typical of the infant with stage 4 BPD (beyond 1 month of life). There is a chronic cough with sputum production. The radiograph shows larger lucencies as well as areas of chronic fibrosis, focal are of atelectasis and marked hyperexpansio Pathologically the alveoli are either overdistended collapsed with few normal areas. The airways still sho squamous metaplasia, but in addition, there hypertrophy of bronchial smooth muscle. Th peripheral airways are narrowed, producing sma airway obstruction. the pulmonary vasculature may als show signs of pulmonary hypertension.

There is a wide spectrum of clinical severity in BP (Table 2) ranging from patients with only mil physiologic and/or radiographic changes and a increased susceptibility to lower respiratory trac infections, to those who require years of hospitalizatio in an intensive care setting, prolonged oxygen therapy a home, and who develop respiratory failure with vira illnesses.

The complications of BDP are frequent, varied, an often serious. Pneumothorax is a frequent complication particularly on the third to fifth days of life as the lung become less stiff and overexpanded on the sam ventilator settings that were barely adequate the da before.

Infants with BPD are particularly susceptible t severe lower respiratory tract infections. This increase susceptibility occurs not only during the acute perio when their normal defense mechanisms ar compromised by prematurity, intubation, oxygen an other unidentified factors, but also during the first fe years of life. Frequently, the infant or young child wit BPD will wheeze, cough and develop cyanosis wit colds. These symptoms may be so severe tha hospitalization is required.Usually these infections are viral but the distinction from bacterial infections is often difficult. Since the chest radiograph usually demonstrates chronic infiltrates and hyperexpansion, acute changes are often difficult to detect. Obtaining a sputum sample is difficult in a one year old but may be done by inducing a cough by applying pressure on the

Table 3
Therapy of BPD

- Nutrition
  - Breast milk or formula
  - Caloric supplements
  - Vitamin supplements
  - Low fluid and sodium loads
  - Iron supplements
- Oxygen
- Assisted ventilation (in hospital)
- Chest physiotherapy
- Diuretics
- Potassium replacement
- Bronchodilators
- Digitalis preparations
- Socialization
- Occupational, physical (and, occasionally, speech) therapists
- Prevention of infection
- Parental education and counseling
- "Team approach"

trachea and catching the sample in the posterior pharynx with a swab. Family history for viral-type illnesses is very important in trying to determine the cause for the increased symptoms in the infant with BPD.

Cor pulmonale and congestive heart failure can be insidious complications in the infant with BPD. They may present as tachypnea with poor feeding and frank cyanosis but frequently a more prolonged history of mild cyanosis, diaphoresis or resting during feedings, and mild increases in cough or wheezing can be elicited. It is quite likely that interstitial edema contributes greatly to the development of these manifestions.

Children with BPD frequently have feeding difficulties. Often vomiting is a persistent problem. The potential causes are multiple; thus, the evaluation must be meticulous. The large amounts of mucus that are coughed up, particularly with infection or percussion and postural drainage, may induce vomiting. Medications such as theophylline, potassium, diuretics, digitalis or over-the-counter cough remedies can be the cause of vomiting. Overfeeding or overconcentrated formulas can also be a problem, or the child may have gastroenteritis or gastroesophageal reflux.

The treatment of BPD consists of meticulous balancing of treatment regimens, side effects of therapeutic agents, and complications (Tables 3 and 4). Oxygen therapy must balance tissue needs with continuing lung damage due to high inspired oxygen. Good nutrition is limited by fluid restriction necessary to prevent interstitial pulmonary edema because of leaky pulmonary capillaries. Use of diuretics is tempered by fluid and electrolyte imbalances. Bronchodilators are helpful but may cause vomiting. Good socialization and integration of the infant into the family is interfered with by the extensive time expenditures required for ongoing care, numerous office visits, recurrent hospitalizations, and the fact that infants with BPD may change their clinical condition rapidly. A viral infection may cause oxygen requirements to increase. Fluid overload may slow the metabolism of theophylline. Vomiting may lead to dehydration and electrolyte imbalance in a patient already on diuretics.

Table 4
Balancing Benefits and Hazards of Treatment

	Too Much	Too Little
Oxygen	↑Lung Damage	Hypoxia Poor growth Vomiting Apnea Cor pulmonale
Fluids	↑Oxygen requirement	Poor nutrition Dehydration
Diuretics	Hypokalemia Hyponatremia Dehydration Toxicity	↑Oxygen requirement
Bronchodilators	Vomiting	↑Work of breathing
Digitalis	Cardiac arrhythmias	Not effective

Good nutrition is crucial for the child with BPD because chronic diseases, per se, interfere with adequate growth. Special formulas and supplements are usually required because of salt and fluid restrictions. Twenty-four and 27 calorie per ounce formulas (as opposed to 20kcal/oz of standard formulas) are available commercially or can be made by adding less water to concentrates of powders. Medium chain triglycerides (MCT oil) or corn oil provide easily digested fat with a very high caloric density of 8kcal per cc. High protein cereal can provide extra calories and thickened feedings without adding a fluid load. Polycose is a sugar polymer that provides carbohydrate without a large osmotic load (2kcal/cc). A combination of these can be used to give as much as 150kcal per 100 cc which may be necessary in some infants whose fluid status is so tenuous that they can only tolerate 100 cc/kg/day but whose metablolic needs are so great that they need 150kcal per kg/day to grow. Extra iron should be added because iron stores cross the placenta mainly in the last two months of gestation. Multivitamins should be given daily. Supplemental vitamin D must be added to the diet of very small infants because their requirements are increased by rapid bone growth and the amount in formulas is predicated on a normal consumption of a liter per day.

Fluid and salt restriction and diuretics are important because these infants have pulmonary capillaries that leak, producing interstitial pulmonary edema. Some infants may be very sensitive to fluids so that a moderate increase in fluids causes respiratory compromise with tachypnea, cyanosis, retractions, rales (crackles) and CO retention. This is usually reversible with diuretics. Many infants require chronic oral diuretics with intermittent parenteral doses.

Bronchodilators are often useful in BPD. The airways appear to be obstructed by muscular spasm as well as edema and mucus. Theophylline compounds not only relieve bronchial smooth muscle spasm but also remove some of the edema fluid by their diuretic effect and may have a salutory effect on ciliary clearance of mucus. The dose of theophlline must be carefully titrated for each patient, using blood levels, because the metabolism of theophylline by infants may vary widely. The sympathomimetic bronchodilators (e.g. isoproterenol) may also be useful in some patients.

Oxygen is often a necessary part of the treatment for infants with BPD (as well as one of the main causes of BPD). Too little oxygen may increase the work of breathing so much that the infant will not be able to gain weight. In addition, persistent hypoxemia predisposes the infant to cor pulmonale. Too much oxygen will further damage the growing lungs and perpetuate the problem. The weaning process for those on oxygen is usually long and slow. Some infants are sent home on oxygen administered by nasal cannula. Blood gases are crucial in monitoring oxygen therapy.

Since these infants are quite sensitive to small changes in oxygen concentration, a change in altitude can potentially cause a setback. Blood gases are helpful in evaluating what effect changes in altitude will have on arterial oxygen tensions. A rule of thumb is that a climb of 1000 feet will lower the alveolar PO_2 by about 6 mmHg (unless there is compensatory hyperventilation) and an increase in the inspired PO_2 by 1% will increase the alveolar PO_2 by roughly the same amount. Thus, an infant being transferred from Tucson at 2500 feet to Flagstaff at 7500 feet woud need about 5% more oxygen. Commercial airplanes are pressurized to about 8000 feet so these infants may not tolerate a long flight without supplemental oxygen.

Chest physiotherapy is an important part of the management of BPD becaue of damage to the normal ciliary mechanism for clearing secretions. Frequently these treatments must be continued by the parents after the baby goes home. Generally, percussion and postural drainage with nasopharyngeal suctioning are scheduled to take place before feedings and to coincide with the maximum effect of bronchodilators.

Cor Pulmonale is a frequent complication of BPD. Eventually the altered dynamics of the right ventricle interfere with left ventricular function and bilateral congestive heart failure may ensue. If congestive heart failure complicates BPD, therapy with diuretics and digitalis preparations may be helpful. The diagnosis of cor pulmonale in an infant with BPD may be difficult. The abnormal breath sounds may mask the loud pulmonic component of the second heart sound, the liver may be pushed down by the hyperexpanded lungs, and overinflation of the lungs may distort the EKG and echocardiographic findings.

Prevention of anemia or polycythemia is another facet of the therapy of BPD. Since iron stores cross the placenta mainly during the ninth month of gestation almost all infants with BPD are iron deficient. The need for frequent blood tests in order to monitor all the aspects of therapy, especially early in the course, oft leads to the need for transfusions. After the first fe weeks, iron supplementation is usually sufficient but t hematocrit must be monitored to ensure an adequa oxygen carrying capacity. Some infants may devel apneic spells if they become anemic. Late in the course BPD, some infants may develop polycythemia if they a chronically hypoxic. This can exacerbate the problems cor pulmonale if the blood viscosity is increased.

Because BPD is a chronic illness that involves ma specialists, a team approach should be adapted from t beginning. The pediatrician, neonatologist, an pulmonologist must work in concert. The nursing sta in the hospital should work to engage the infant i appropriate socialization. Occupational and physic therapists should become involved as soon as the infa is stable. After discharge, visiting nurses and therapist can be very helpful to the parents by making suggestion or arranging for services. Social workers can ease som of the financial problems and provide counselling for th parents.

The parents should be encouraged, early in the cours of the disease, to be involved in the care of their infan They should understand all aspects of their infant's car and become facile with administering medications, ches physiotherapy, as well as the operation of suctio machines or oxygen equipment if they are going to us these at home. They should learn to recognize dange signs such as duskiness and learn to distinguish behavio disorders from medical problems. They shoul understand the importance of avoiding respirator infections by minimizing the contact of their infant wit those with colds. Taking home an infant with BPD ofte causes considerable disruption of the family unit. Th stress of the initial, often prolonged hospitalization with its concomitant financial worries and concern about the health of their child are often compounded b the necessities of day to day care with frequen treatments, medications and doctor visits and th possibility of rehospitalization. Behavior problems i siblings may develop as well.

The prognosis for children with moderate to severe BPD is still not known because of its relative "newness." The long term studies of children born in the 1960s are quite encouraging with many of these children showing near normal pulmonary function after five to ten years.[21,22] However, these children tended to have milder forms of the disease. With smaller and more premature infants surviving in the 1970s there is the possibility that these children will not do as well. However, there is some suggestion that new techniques such as nasal CPAP and refinements in respirator managment have decreased the incidence and severity of BPD. A better understanding of the mechanisms of oxygen and mechanical injury to the lungs will allow us to prevent damage by more judicious respiratory therapy. Even more preferable, would be the prevention of prematuare births altogether.

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Seminars in Dermatology

Scleredema:
A Review of this Disorder and Presentation of a Case

David L. Graham, M.D.
Andrew S. Tedesco, M.D.
Robert Allen Schwartz, M.D., M.P.H.

Editor:
Robert A. Schwartz, M.D.

Scleredema is an uncommon disorder often confused with localized scleroderma (morphea) by those unfamiliar with it. However, scleredema is a clearly distinct process both clinically and microscopically. It appears to be a disorder characterized by hyaluronic acid deposition in the dermis. Its association with diabetes mellitus is also of interest. We present a 53 year old man with scleredema and diabetes mellitus in whom the scleredema presented as a cellulitis of the back.

Case Report

A 53-year-old Lebanese man came to the Life Support Unit at the Tucson VA Medical Center in April, 1979 for evaluation of fever. He had stable adult onset diabetes mellitus controlled by diet and Lente insulin. There was also a history of angina pectoris controlled by propranolol. The patient was in his usual state of health until three weeks prior to admission when he was treated with penicillin for a streptococcal pharyngitis. He was otherwise well until the day of admission when

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From: Department of Internal Medicine, Section of Dermatology, Univ. of Arizona Health Sciences Center, Tucson, AZ 85724. Dr. Graham is now with the Department of Dermatology, Stanford University. Reprint requests to Dr. Graham.

he noted sudden onset of fever, chills, nausea and lethargy. He denied any awareness of skin changes, including the indurated skin of his back noted in physical examination. There was no history of vomiting, cough, polyuria, chest pain, arthralgias, dysphagia or muscle weakness.

Initial evaluation revealed a moderately toxic appearing man with temperature 104° F; other vital signs were normal. The skin was warm and moist with a 60 x 60 cm thickened, brawny, tan, indurated, nonpitting plaque covered by nonpainful, nonpruritic, diffuse erythema. Laboratory examination revealed a white blood count of 5,5000/cu mm, with 64% polymorphonuclear leukocytes, 1% bandforms, 15% lymphocytes, 11% monocytes, and 1% eosinophils. Hemoglobin was 14.0g%. Urinalysis was negative for glucose and ketones, had 2-3 WBC/HPF and occasional bacteria.

Serum glucose was 257 mg%; electrolytes, creatinine and BUN were normal. CSF was unrevealing. The chest x-ray showed eventration of the right diaphragm, but was otherwise normal. The electrocardiogram demonstrated normal sinus rhythm, left axis deviation and nonspecific ST-T wave abnormalities. Sputum gram stain showed mixed flora. Blood cultures were drawn.

A tentative diagnosis of acute pyogenic cellulitis was made and the patient placed on methicillin, 1 gm, I.V. every 6 hours. Other etiologies considered were scleroderma, eosinophilic fasciitis, dermatomyositis, and scleredema. Preadmission medications of Lente insulin 40 U. every a.m. and 10 U. at 4 p.m. were continued along with propranolol.

Tests for fluorescent antinuclear antibody (FANA), rheumatoid factor and cyroglobulins were negative. A serum protein electophoresis was normal. Antistreptolysin titer was less than 100 STZ units (normal, up to 100). Urine and multiple blood cultures were unrevealing. Biopsies of two skin sites were obtained. Histologic examination of the patient's tissue revealed thickening of the dermis with separation of the collagen bundles. Deposition of hyaluronic acid within the separations was shown with colloidal iron stain.

After blood culture results were obtained the following day, methicillin was discontinued. The patient continued to defervesce and other symptoms remitted. Erythema cleared over the plaque. The patient had one episode of angina treated with nitroglycerin.

A discharge diagnosis of acute viral syndrome, complicating angina, diabetes, and scleredema was made. The patient was sent home asymptomatic on the same regimen of propranolol and Lente insulin.

Comment

Scleredema was originally described by Buschke in 1900. It is a disease of unknown etiology frequently following a viral or bacterial infection by several days to weeks. There is often a prodrome of myalgias and general malaise followed by the relatively acute onset of localized induration of the skin usually of the neck, back and trunk. In general, it spares the hands and feet. The induration is nonpitting, firm, nontender with a wa superficial appearance not unlike scleroderma. In fa the initial impression is frequently that of localiz scleroderma (morphea) or of eosinophilic fasciitis. It m also be confused with cellulitis, which was consideration in our case.

Visceral involvement is uncommon in sclereden However, there have been reports of involvement of t tongue, skeletal muscle, pleura, pericardium, heart, liv and spleen. Leinwand[3] reported an autopsy case of a year-old woman who apparently died of viscer involvement and organ failure secondary to edema the liver, heart and spleen as well as pleural a pericardial effusions. He did not, however, demonstra mucopolysaccharides in the involved organs. Valle[4] al feels that scleredema is a systemic disease and he reported four cases in which there was widespre organ system involvement in each but no viscer biopsies were done.

Robinow[5] in 1963 reported two cases of sclereden "adultorum" in children under the age of 13, and or month later, Greenberg et al[6] reported four more cas of scleredema in children; both observed th approximately one third of the cases occur in childre Jablonska[7] stresses that the term "adultorum" should dropped and scleredema alone should be used; we conc with this opinion. The usual time course had been said be resolution within six months with few cases lastir up to several years. Curtis et al[8] presented eight case reviewed the literature for 215 more cases and noted th incidence of persistence of symptoms to be 33% in adul and 21% in children in the 109 patients that we followed for two years or longer.

Scleredema and diabetes mellitus of the maturi onset type have been noted together in numero patients. Fleischmajer et al[1] described eight cases scleredema all associated with obesity and maturi onset diabetes mellitus. Most of his patients were insul resistant as well. Margolis[2] reported a case of sclereden and diabetes mellitus and differentiated this syndron from scleredema. He feels that scleredema followin diabetes occurs after longstanding diabetes witho prodrome of preceding infection whereas sclereden usually follows a prodrome suggestive of an infectio

Microscopic examination reveals a thickened derm with separation of the collagen bundles and th demonstration with special stain of hyaluronic aci between the bundles of collagen. Hyaluronic acid is a acid mucopolysaccharide which visualizes well wit colloidal iron stain.[9] Some cases of persisten longstanding scleredema have been termed "scleredem diutinum"; these cases are said to sometime lack th hyaluronic acid in the tissue specimens.[10]

The differential diagnosis of scleredema include eosinophilic fasciitis[11] and localized scleroderm (morphea) (Table 1). Occasionally, progeria, trichinosi graft vs. host reactions, systemic amyloidosis, porphyri cutanea tarda, polyvinyl chloride disease, lichen sclerosi

	Eosinophilic Fasciitis	Localized Scleroderma Morphea	Scleredema
tiology	Preceding strenuous activity?	Preceding trauma?	Preceding streptococcal infection implicated
lanifestations	Spares face. Extremities involved. "Bound down" appearance Raynaud's phenomena-absent	Variety of lesions - guttate plaque, generalized, linear waxy-shiny lesions, lilac colored border Raynaud's rarely present	Pale symmetric induration of large areas of skin. Painless swelling, firm non-pitting induration Raynaud's absent
ncidence	Age 4-60 male: female: 2:1	any age especially young (20-40 yrs.) and children Females predominate 3:1	2/3 are female 50% adults
aboratory	1. Eosinophilia 2. Hypergammaglobulinemia 3. ESR often↑	1. CBC - WNL 2. ESR - WNL 80% of time 3. Serum proteins - WNL	1. ASO titer ↑ especially in children 2. ESR - WNL or slightly ↑ 3. Serum proteins - usually WNL
listopathology	A. Dermis 1. Early - inflammatory cells, including eosinophils 2. Late - dermal changes of scleroderma B. Deep Fascia 1. Collagenous hypertrophy 2. Infiltrate-plasma cells, lymphocytes, eosinophils	A. Dermis 1. Early - packed, thick collagen bundles - inflammatory infiltrate more pronounced in sub-cutaneous fat 2. Late - dermal collagen closely packed. Inflammatory infiltrate nearly gone 3. Sweat glands appear well within the dermis instead of at the dermal-subcutaneous jct, since most of sub-cutaneous fat is replaced by newly formed collagen	A. Dermis 1. Three times thicker than normal 2. Separation of collgen resulting in fenestration 3. Subcutaneous fat replaced by dermal collagen bundles 4. Dermal deposition of hyaluronic acid may be demonstrable with special stains
reatment	Possibly systemic steroid responsiveness	1. Intralesional steroids 2. Systemic steroids - not indicated 3. penicillamine: results variable 4. diphenylhydantoin: results variable	Many forms 1. systemic steroids 2. pituitary and thyroid extract inconsistent reponse 3. reassurance

et atrophicus, and phenylketonuria have associated sclerodermoid changes; these disorders are not likely to be confused with scleredema clinically. Bleomycin toxicity has also been linked with cutaneous sclerodermoid changes,[12] as has occupational trauma, as in the use of pneumatic hammers.[13] — both are easily distinguished from scleredema. With scleroderma, the histologic picture differs from scleredema; in the former, collagen bundles are more closely packed as well as thickened. In eosinophilic fasciitis the dermis and epidermis are usually normal, but the deep fascia is involved with an infiltrate containing eosinophils, plasma cells, histiocytes and lymphocytes.

Acknowledgment

This patient was presented at the Tucson Veterans Administration Hospital Clinical Management Conference by Drs. Andrew S. Tedesco, Robert A. Schwartz and Jamie Kroeger. Drs. Jamie Kroeger and David Bowman referred the patient to us.

The authors wish to thank Ms. Maria Garlant for her secretarial assistance.

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Seminars in
Endocrinology and
Metabolism

Why Control Blood Glucose Values?

Fred D. Hofeldt, M.D., F.A.C.P.

Editor:
Stephen E. Dippe, M.D.

The short term beneficial effects of insulin on hyperglycemia are well recognized and undisputed.[1] These include prevention of diabetic ketoacidosis, hyperosmolar coma and dehydration, correction of electrolyte imbalance, normalization of plasma lipids, enhancement of polymorphonuclear leukocyte and lymphocyte function, better wound healing and enhancement of the quality of the diabetic life with improvement of fatigue, symptomatic polyuric states and vision. In the past, great controversy has existed regarding similar beneficial effects with control of blood glucose on the chronic complications of the disease. However, today hyperglycemia promotes the development and progression of the chronic complications[2 8] Let us review the data which would support this statement. Numerous studies in diabetic animal models provide the greatest evidence of the harmful effects of hyperglycemia on the triopathy of diabetes (retinopathy, nephropathy and neuropathy).

In regard to the nephropathy of diabetes, post-transplantation data from diabetic animals and patients have shown that there is early hyalinization of both the afferent and efferent arterioles. With time these kidneys develop glomerulosclerosis or typical Kimmelstiel-Wilson renal lesions. Sensitive measurements of albuminuria by radioimmunoassay technique has established that early in their disease insulin dependent diabetics may have a significant protein loss with normal urinary excretion of beta-2 microglobulin (this measures tubular protein reabsorptive function).[9] When seven diabetic patients with evidence of albuminuria were treated by a three-day constant insulin infusion, urinary excretion of beta-2 microglobulin remained normal whereas urinary albumin losses diminished; supporting the rapid reversibility of the albuminuria with the establishment of control of the hyperglycemi
Within two weeks following the acute precipitation
diabetes mellitus in the experimental rat, albuminuri
appears and can be cleared within two months followin
islet cell transplantation.[10] Numerous transplantatio
studies confirm improvement in renal status in islet ce
transplant recipients.[11 13] When two diabetic kidney
were inadvertently transplanted into a normal recipien
the diabetic changes in the transplanted kidney
returned to normal within six months.[14] A transplan
recipient who underwent bilateral nephrectomy becaus
of obstructive nephropathy when transplanted a norm
kidney (both donor and recipient had no history
diabetes) developed steroid induced hyperglycemi
which caused typical diabetic nephropathy changes i
the transplanted kidney.[15] Thus both animal and huma
studies show the development and reversibility of th
renal diabetic lesions and its alteration with control o
hyperglycemia.

In regard to the retinopathy of diabetes, studie
performed in dogs by Dr. Bloodworth[16] show that wit
the diabetic state there is progressive development o
diabetic microangiopathy changes in the retina. Whe
the diabetes is treated with only one shot of insulin
day, progression of the retinopathy is evident. However
when glucose values are normalized by two insuli
injections per day, there is an arrest and reversal of th
diabetic retinopathy process. A Japanese study[1]
evaluated the course of diabetes in 356 patients over si
years and noted a correlation between absence of ne
lesions and reversal of old lesions in regard to goo
diabetic control. In 52 patients reported by Job,[18] ther
was a positive correlation with the progression o
diabetic retinopathy and hyperglycemia whereas
reversal and lack of progression of the retinopathy wa
noted in those diabetics with good control. Critics of thi
study point out that this group of patients is too small t
make valid statistical comparisons, that studies wer
preformed in only one diabetic eye in regard to th
observed progressive changes and that there wa
considerable crossover between treatment group
which invalidates the statistical conclusion of this study
Using a new technique of fluorophotometry, it has bee
possible to detect capillary leakage in diabetic patient
who have normal ophthalmopathy examination an
fluorescein angiography. In 77 diabetic patients[19] s
studied (negative opthalmic examinations and negativ
fluorescein angiography), increased capillary leakage o
fluorescein was noted and the abnormalities wer
postively correlated with the degree of diabetic contro
and duration of diabetes. Interestingly, similar studies in animals using this technique have shown that the fluorscein leakage in diabetic rats reversed to normal within two weeks following islet cell transplantation.[2

Studies regarding control and diabetic neuropathy have been more conclusive in animal models than in patients. In animals there is a rapid decrease in nerve conduction velocities within two weeks following the

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From: University of Colorado Medical Center, Denver, Colorado. (Dr. Hofeldt, Associate Clinical Professor).

onset of diabetes mellitus. This abnormality is not corrected with insulin unless the insulin is given as twice daily injections to strictly normalize the blood sugar (less the 100 mg/dl fasting blood glucose) in association with frequent hypoglycemic reactions.[21] Only when this degree of control is obtained will there be definite evidence of reversal of the nerve conduction abnormalities. In these diabetic rats, the abnormality in nerve conduction may be reversed by supplementing the rats' standard diet with myoinositol.[22] When the diet is so supplemented, the nerve conduction studies returned to normal despite the presence of hyperglycemia. These studies implicate the loss of intracellular myoinositol to the diabetic nerve lesion. The findings of elevated plasma myoinositol and elevated urinary excrections of myoinositol in diabetic human subjects would suggest perhaps myoinositol suggests a role in the metabolic neuropathy. However, it has yet to be shown that myoinositol supplementation in man plays a major beneficial role in treatment. When exposed to hyperglycemia, the diabetic nerve cell increases metabolic substrate along the sorbitol pathway corresponding to increased enzyme activity of aldose reductase and sorbitol dehydrongenase within the specific tissues that possess these enzymes. Tissues noted for the accumulation of sorbitol and fructose have been the lens, nerve tissue and the aorta. Despite vast knowledge of this metabolic pathway, its role in diabetic neuopathy is speculative but clinically it may be implicated in the acute reversible hyperglycemic neuropathy. Diabetic peripheral neuropathy is one of the most difficult diabetic disorders that the clinician will face when treating the complications of diabetes. Although in the laboratory model it may be reversed with good control, such response has not been dramatic in patients. Although a number of therapeutic modalities have been tried, they are generally unsuccessful in the managment of this condition. Graf[23] recently reported that in 20 untreated, newly diagnosed diabetic patients there was a positive correlation between motor nerve conduction velocity abnormalities and the fasting blood glucose values. Similar correlation was noted in regard to the level of hemoglobin A1C. Hemoglobin A1C is a nonspecific tissue indicator of glycoprotein formation and may serve as a marker which mirrors the microvascular glycoprotein accumulation seen in the diabetic microvascular disease. This red blood cell hemoglobin may conceptually be viewed as a "sugar coated doughnut" (comment by Dr. George Cahill).

Studies of diabetic microvascular changes in muscle capillary basement membranes have initially been confusing and misleading. Premature conclusions suggested that good control did not prevent such complications. These early studies noted that diabetic patients had a characteristic thickening of muscle capillary basement membranes and these measurements could predict a "pre-diabetic condition". Based on numerous studies, the conclusions now accepted are that the capillary basement membrane thickening is associated with poor control and longstanding duration of the diabetes. The lack of capillary basement membrane thickenings noted in newly diagnosed juvenile onset diabetics and the appearance of capillary basement membrane thickening in poorly controlled juvenile onset diabetics after two to five years of the disease supports the acquired nature of this observation.[24] Dr. Siperstein reported that patients who had secondary forms of diabetes did not have abnormal capillary basement membranes. Subsequent studies by Dr. Raskin[5] on even some of Dr. Siperstein's original patients with secondary diabetes and hyperglycemia have shown that after a mean interval of five years there is increased basement membrane formation in these patients. It is now evident that secondary forms of diabetes mellitus with hyperglycemia are associated with the triopathy and that these patients are not spared the long term complications that are seen in idiopathic diabetes mellitus. These observations implicate hyperglycemia in the acquired microvascular complications rather than as an independent genetic trait. In regard to children with newly diagnosed diabetes mellitus, Dr. Jackson has shown that if these children are strictly controlled (ie, normal fasting blood glucose values and plasma glucose profiles with no glycosuria) then these patients may have their diabetes for up to 20 years and yet not show progression in basement membrane thickening, as compared to other juvenile onset diabetics who are poorly controlled.[24] These observations provide the major evidence in man that the glycoprotein abnormalities in basement membrane formation are related to hyperglycemia per se. Even more recently the data of the controversial UDGP study have supported the thesis that poor control and diabetic cardiovascluar complications are related.[25]

The macrovascular complications seen in peripheral vessels (pelvis, legs, and cerebral circulation) and coronary arteries of diabetic patients are a different story. Although there may be some decrease in the incidence of this process with regard to control of the hyperglycemia,[7] the macrovascular disease in diabetic patients is multifactoral. This process involves tissue reaction to intimal injury with smooth muscle cell proliferation, formation of large amounts of connective tissue matrix, deposition of lipids both within cells and in the connective tissue. Early lesions include the fatty streak and fibrous plaque with the late lesion being a fibrous plaque resulting from degenrative changes, hemorrhage, calcification, necrosis, thrombosis and calcification.[25] In diabetic patients, a major factor influencing this complication may be their inherent increased aging. Dr. Benditt[27] in Seattle has shown a decreased life span of fibroblasts from diabetic patients carried in tissue culture versus normal controls. Likewise, collagen dating[28] of diabetic collagen shows considerably older collagen than in age adjusted, nondiabetic control subjects. This data suggests an inherent aging factor in diabetic patients independent of the metabolic alterations.

Likewise, since these large vessles possess the metabolic enzymes of the sorbitol pathway,[29] hyperglycemia promotes accumulation of osmolar metabolites which initiate intimal edema causing poor nutrient diffusion and uptake by the aorta and large blood vessels. This may lead to intimal damage. Decreased oxygen diffusion occurs across these osmolar loaded cells which further compounds the ischemia. With intimal injury, the increased platelet adhesiveness characteristic of the diabetic state provokes thrombosis and clot formation. In a large study of 4,400 patients in France by Dr. Pirart,[7] the presence of hyperglycemia was correlated positively with the triopathy of diabetes mellitus but did not show a good correlation with the macrovascular disease of diabetes mellitus. These data would suggest that in our search for the etiological factors of diabetic macrovascular disease, hyperglycemia, arterial intimal wall metabolism, injury and repair; arterial smooth muscle cell proliferation; hormonal agents; serum lipids; platelet function and aging require careful consideration.

Hyperglycemia begets both acute and chronic complications of diabetes and hence over the "short and long run" adversely affects the quality of life for diabetic patients. However, despite this knowledge, we lack practical methods for "strict" control or normalization of blood glucose levels.

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Comments

Yes, Control Blood Glucose Values

Stephen E. Dippe, M.D., F.A.C.P.

For years there has been a "tug-of-war" between diabetologists whether or not "excellent" diabetic control helps to prevent, or at least lessen the complications of diabetes mellitus. The chronic complications of diabetes are frequently devastating, causing blindness, kidney failure and impaired nerve function. There were those physicians who genuinely believed that controlling diabetes had little or no effect on these complications. They believed that a more liberal approach allowed for a more normal existence. Now we are hearing many experts encouraging practitioners to attempt to control diabetes as best as possible within the limitations of the patient's willingness to undergo good control.

Dr. Hofeldt's article gives considerable evidence to support the concept that elevated blood glucose levels adversely affect the chronic complications of diabetes. He has systematically presented the data which appear to be highly convincing. In fact, the American Diabetes Association in 1976 officially supported optimal control of diabetes and stated, "Current clinical and experimental data clearly demonstrated that optimal regulation of blood glucose levels should be achieved in the treatment of diabetes, particularly in young and middle-aged individuals who are at greatest risk of developing microvascular complications."[1]

To obtain optimal diabetic control requires the patient to become very knowledgeable about diabetes. He must also feel comfortable and at ease with his diabetes. Frequently, it will require the use of both intermediate-acting insulins and short-acting insulins in combination and may require two injections per day. The patient must have a good understanding of the need for dietary control, exercise and other factors that contribute to good diabetic control.

A major question yet to be completely answered is, "What is optimal diabetic control?" As of today, we have no clear-cut answer but to obtain glucose values as near normal as possible taking into consideration a number of factors about the patient, such as his age, occupation, work habits, mental, physical and emotional status as well as the patient's motivation, compliance and determination for better control. The patient's knowledge about acute complications is absolutely essential partcularly that of hypoglycemia. Personally, I believe it is important to honestly evaluate if the patient will adhere to such a program or if he is capable of adhering to such a program and if it would cause any deterioration in social, economic or emotional stability. Thus, many factors must be considered in determining how tight the patient's diabetic control should be.

It is a pleasure to see Dr. Hofeldt go through the literature in a very clear fashion giving the evidence as accumulated on how neuropathy, retinopathy and nephropathy are related to elevated blood glucose levels. The evidence is in and supports good control. Finally, we are seeing fewer "doubting Thomases" and we are seeing fewer individuals who are willing to dispute the weight of evidence that is presented.

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From: Scottsdale Memorial Hosptial, Family Practice Center, 7301 East forth Street, Ste 22, Scottsdale, AZ 85251.

Seminars in
Gastroenterology
and Liver Disease

Pancreatic and Biliary Complications of Side-to-Side Choledochoduodenostomy

George E. Burdick, M.D.

Editors:
George Burdick, M.D.
Stephen Glouberman, M.D.
Leon Rigberg, M.D.

Over the past few years the author has seen a number of patients who were referred for evaluation of recurrent cholangitis and/or pancreatitis post side-to-side choledochoduodenostomy. Many of these patients were found to have food particles and other debris in the intrapancreatic portion of the biliary tree. These particles were the cause of their clinical problems. A typical case is presented below.

Case History

In April of 1976 a fifty-six year old Caucasian female presented to Doctor's Hospital with a history of recurrent epigastric pain and abnormal liver profile studies. She had been having recurrent pain off and on for approximately eight years. In March of 1976 she presented with a very high SGOT which dramatically dropped to normal over a couple of days. At other times the patient had moderate elevations of serum amylase and was thought to have recurrent pancreatitis. A previous intravenous cholangiogram had been interpreted as normal.

The patient had some calcifications thought to be in the pancreas which were demonstrated on a flat plate of the abdomen. She had a history of moderate alcohol consumption which had been assumed to be related to her episodes of pancreatitis. Approximately ten years previously the patient had a cholecystectomy. Subsequently, the patient had a choledochoduodenostomy after a common duct exploration for stones an distal bile duct stricture.

Physical Examination

Physical examination revealed a pleasant Caucasia female who was afebrile at the time of examination HEENT was normal with no evidence of scleral icterus Heart and lungs were within normal limits. Examinatio of the abdomen revealed a well-healed right uppe quadrant subcostal incision. The liver was palpable tw finger-breadths below the right costal margin an slightly tender. The left lobe was also thought to b slightly enlarged. No other masses or abnormalitie were noted. There were no other areas of significan tenderness. The patient had no periphera manifestations of liver disease.

Hospital Course

The patient was placed on antibiotic therapy o admission and rapidly became afebrile. The admissio liver profile demonstrated a bilirubin of 1.2 mg%, SGO of 137 U per 100 ml and an alkaline phosphatase of 51 IU. A serum amylase was not performed initially. Th patient's white blood count was 8,900 with a prominen shift to the left; the hematocrit was 35%. After initia stabilization, gastroenterology consultation wa obtained and an endoscopic retrograd cholangiopancreatography was recommended. ERC was preformed on the 23rd of April, 1976 an demonstrated the findings in Figure 1. Several filling defects were demonstrated in the intrapancreati

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From: 1010 E. McDowell Road, Ste 204, Phoenix, AZ 85006.

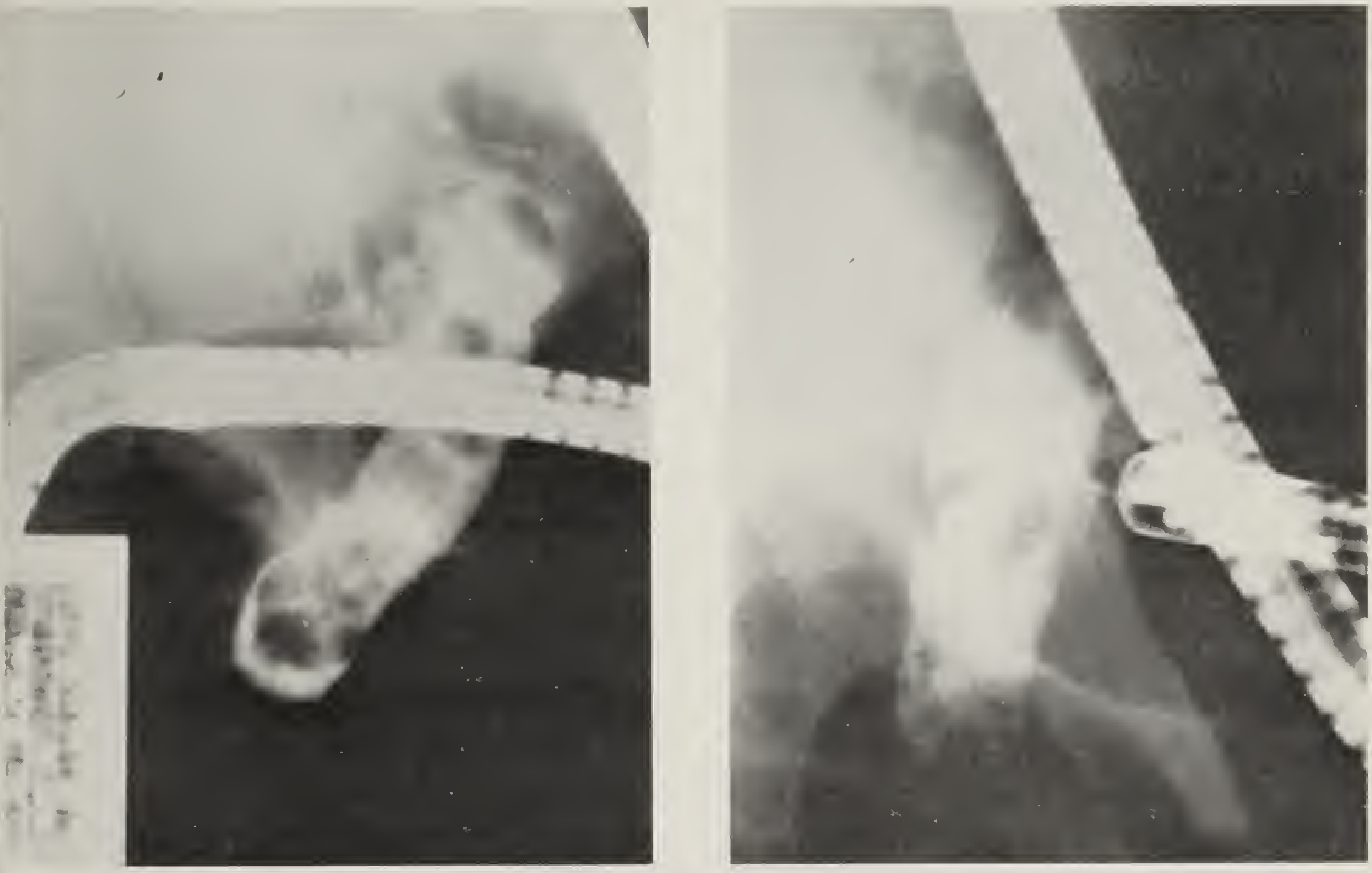

Figure 1

1A - Filling of common bile duct through papilla of Vater demonstrating multiple filling defects.

1B - Filling of bile duct through side-to-side choledochal anastomosis.

portion of the common bile duct. A recommendation was made that the patient undergo Roux-en-Y choledochojejunostomy. Surgery was performed the following day. The patient had an uneventful postoperative course. It is now over three years since the patient's surgery and she has been asymptomatic since that time. Her liver profile has returned to normal.

Discussion

In general, physicians are unaware that recurrent cholangitis and/or pancreatitis are relatively frequent complications of side-to-side choledochoduodenal anastomoses. Endoscopic retrograde cholangiopancreatography has the unique ability to evaluate patients with recurrent pancreatitis and/or cholangitis. We have seen several patients with similar clinical histories and findings over the past six years because of the large volume of ERCP's performed in our practice.

The frequency of these complications, ie, the percentage of patients with side-to-side choledochoduodenostomy who develop cholangitis or pancreatitis, may be quite low. The author is unaware of a reliable statistic in this regard.

In evaluating patients with side-to-side choledochoduodenostomy one has to consider that the pancreatitis and/or cholangitis may be occurring for other reasons. Endoscopic retrograde cholangiopancreatography should be performed to demonstrate:

1. Food particles, stones or other debris in the intrapancreatic portion of the biliary tree, and
2. Otherwise normal pancreatic duct and biliary tree

General evaluation of the metabolic and drug induced causes of pancreatitis should also be undertaken as described in a previous article.[1]

Once the diagnosis of "succotash cholangitis" seems secure, a procedure designed to remove the opening of the bile duct from the path of food particles is curative. Examples of surgical procedures to accomplish this goal are:

1. Roux-en-Y choledochojejunostomy
2. Billroth II gastrojejunostomy

The importance of biliary and pancreatic complications of side-to-side choledochoduodenostomy has only recently become appreciated, even by gastroenterologists and surgeons. "Succotash in the biliary tree" should be a part of the differential diagnosis in a patient who presents with pancreatitis or cholangitis and a history of previous biliary tract surgery.

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Neurology

Transsphenoidal Approach To the Pituitary

Lewis J. Brown, M.D.

Editors:
Betty F. Ball, M.D.
Barry Hendin, M.D.
Lawrence Z. Stern

The transsphenoidal approach to the pituitary gland is not a new operation. Indeed, if Harvey Cushing were living, he undoubtedly would express great surprise over the present-day interest in the procedure. For Cushing, it would mean a return to the early 1900's when he performed the operation on numerous occasions. Historically speaking, however, he was not the first surgeon to approach lesions of the pituitary by this route. The impetus came from Germany in 1907 by Schloffer[1] with later improvements by Halstead.[2] Oscar Hirsch, a Viennese (and later) Boston surgeon published his initial experience in 1910 and continued with his interest in the operation for the next 35 years.[3,4] He, more than anyone else, prevented the operation from becoming obsolete. In later years, he received support in these efforts from another Boston colleague, Hannibal Hamlin.[5]

Very early in his career, Cushing become interested in the physiology and pathology of the pituitary. To observe the changes produced by absence of the gland, he experimentally removed same from dogs in the laboratory. These operations were done via the subfrontal approach. In 1902, he operated on several patients with large pituitary tumors but could accomplish nothing more than a decompression craniectomy to preserve vision. With these experiences, he became convinced that the direct intracranial exposure of such lesions was too limited to allow their successful removal. He then directed his attention to the Schloffer technique and with some modifications began a series of transsphenoidal operations in 1909. By 1912, he had performed 31 such procedures out of a total of 43 pituitary operations and used these cases as the basis for his classical monograph, "The Pituitary Gland and Its Disorders."[6] In the last chapter of his book, Cushing describes the then-performed operation in his usual detailed manner. The account is fascinating to read for one immediately recognizes that, except for the absence of present-day instrumentation, the described procedure is exactly the same as that which is being carried out today.

Cushing continued to operate on pituitary tumors via this method until the mid-20's. At this juncture in his career, he developed a renewed interest in the subfrontal approach. There were several reasons for the change, a major one being the desire to see the optic nerves and chiasm in cases of large tumors. These principals were expounded by Frazier[7] in 1919 and along with rapid improvements in anesthesia and intracranial surgery in general, the trend became obvious. Cushing himself later admitted that he thought visual recovery was better if the optic apparatus could be viewed directly.[8]

By the time he completed his illustrious career in 1932, Cushing had performed 338 pituitary operations; 227 or 66% were done transsphenoidally, a fact not well recognized even in the neurosurgical community. His mortality rate for the entire series was an unbelievable 6.3% and an even better 5.8% for those performed transsphenoidally.[9]

Credit for renewed neurosurgical interest in the procedure goes to Guiot of Paris.[10, 11] In 1958 he wrote of his experiences with the operation using intraoperative fluoroscopy as an adjunct. This interest was further extended by Hardy who almost single handedly made neurosurgeons aware of this "new"

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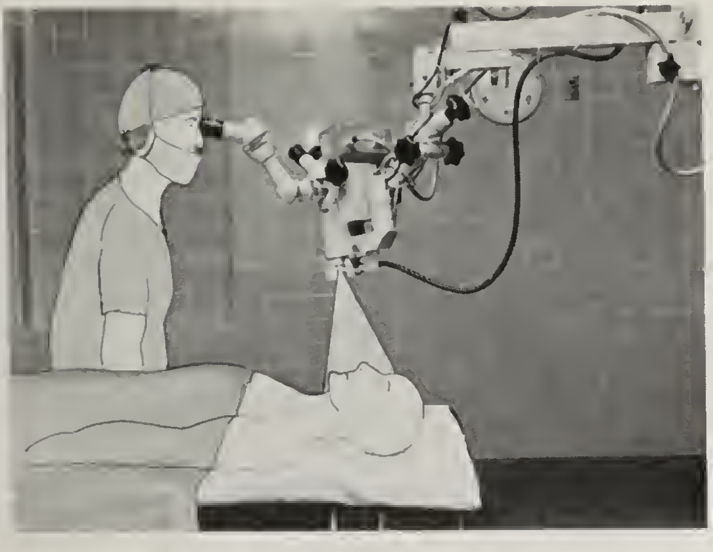

Figure 1
Semindiagramatic illustration of operating microscope in use.

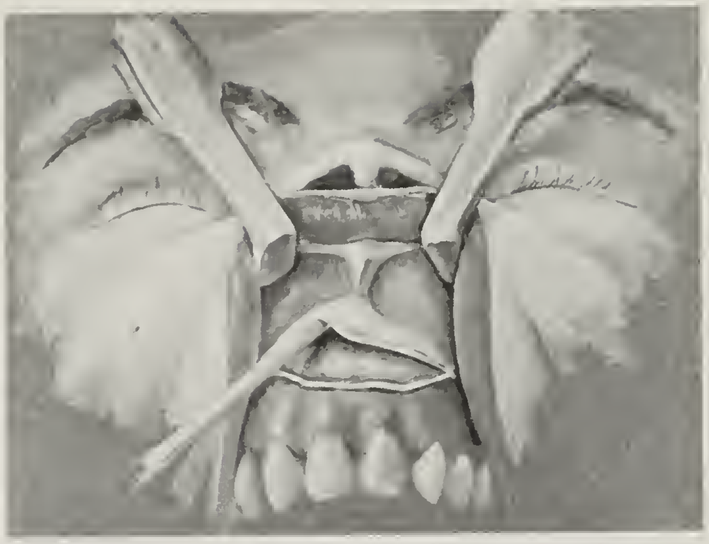

Figure 3
Upper sublabial incision initiates operation.

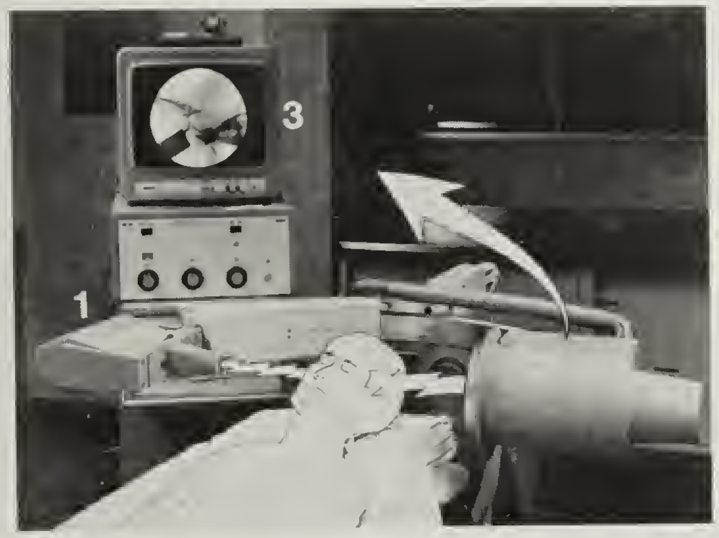

Figure 2
Semidiagramatic illustration of C-arm fluoroscopy in use.

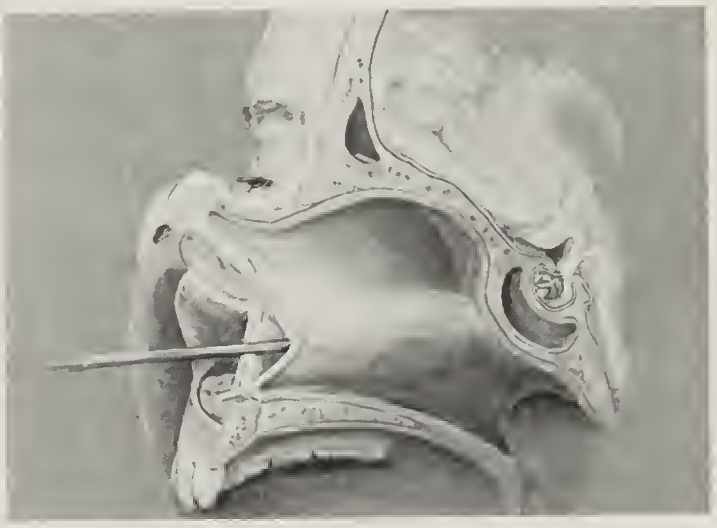

Figure 4
Submucosal dissection to sphenoid sinus.

operation.[12]

Dandy[13] criticized the operation because "at best the actual area of exposure of the hypophysis is scarcely larger than the circumference of a lead pencil." Today we counter this statement with the operating microscope, a precise instrument that allows the surgeon to work in small, narrow areas under great magnification and with excellent lighting (Figure 1). The additional use of intraoperative fluoroscopy aids the operator from straying beyond the limits of the sella or tumor capsule (Figure 2). Harvey Cushing did not have these two adjuncts available to him but they are a necessary part of the modern operating theater for the completion of a safe and successful transsphenoidal operation.

Recent major advances out of the operating room have further promoted interest in this subject in general. Development of the radioimmunoassay technique has allowed the endocrinologist to make biochemical identification of small pituitary adenomas frequently before radiographic evidence of sellar pathology occurs. Although routine skull x-rays may be normal, minute changes in the sellar floor can frequently be identified with the hypocycloidal movement of the polytome. Finally, computed brain scanning (CAT) will visualize some pituitary lesions and will depict their relationship to the base of the brain and third ventricle.

Technique of Surgery

Following induction of anesthesia, the patient is placed in a semi-sitting position with slight head flexion. A mobile C-arm radiofluoroscopic image intensifier is positioned to provide lateral viewing of the cranium on the television monitor. After a standard nasal prep, the upper lip and gingiva, as well as the nasal septum, are infiltrated with a local anesthetic containing Epinephrine. A small area on the right thigh is prepared for the taking of a graft at the completion of the procedure.

A horizontal incision is made through the previously infiltrated upper gingiva (Figure 3). Submucosal exposure of the septum and nares proceeds to the face of the sphenoid (Figure 4). The posterior one-third of the

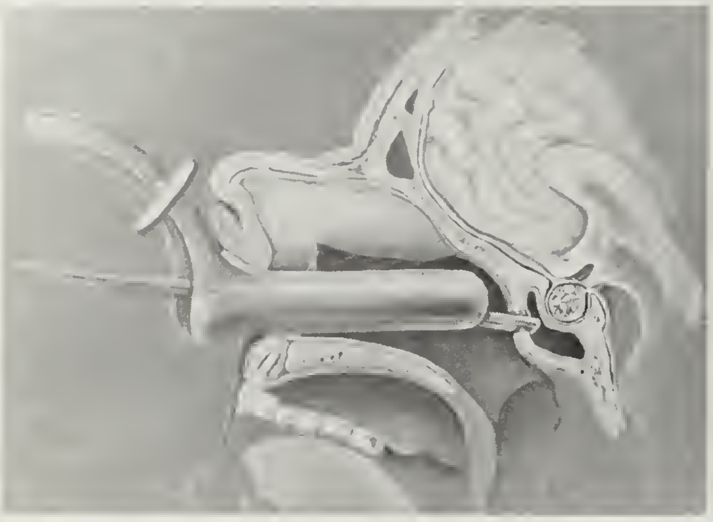

Figure 5
Sellar floor removal and dural exposure.

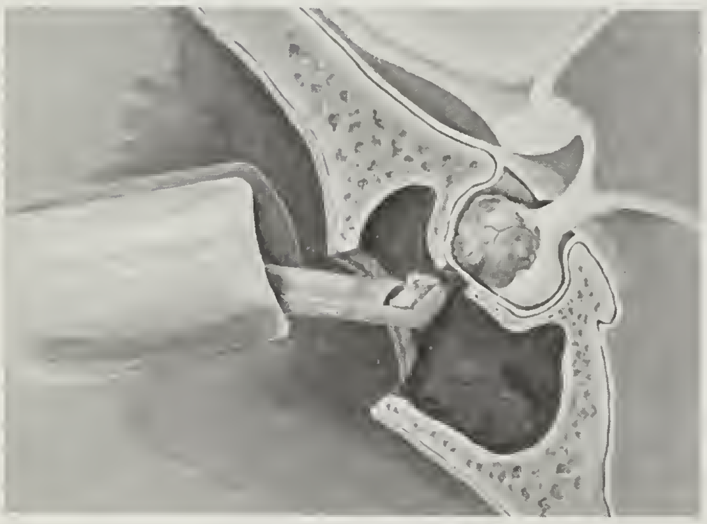

Figure 6
Sellar floor removal and dural exposure.

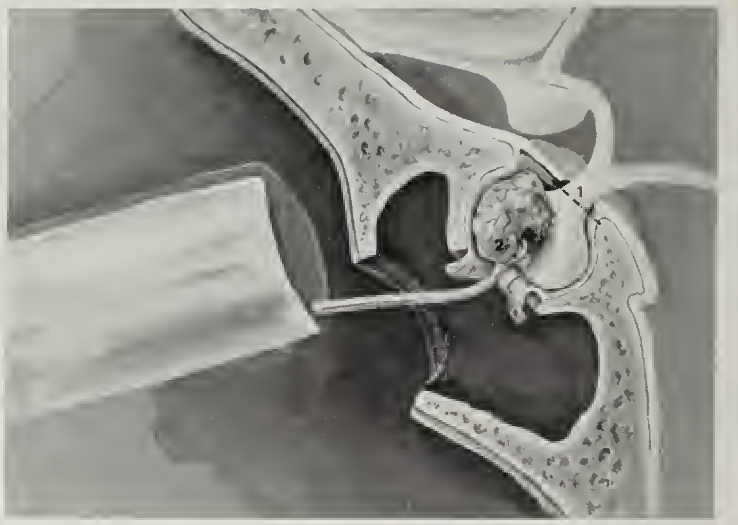

Figure 7
Total gland removal 1) or selective tumor excision 2).

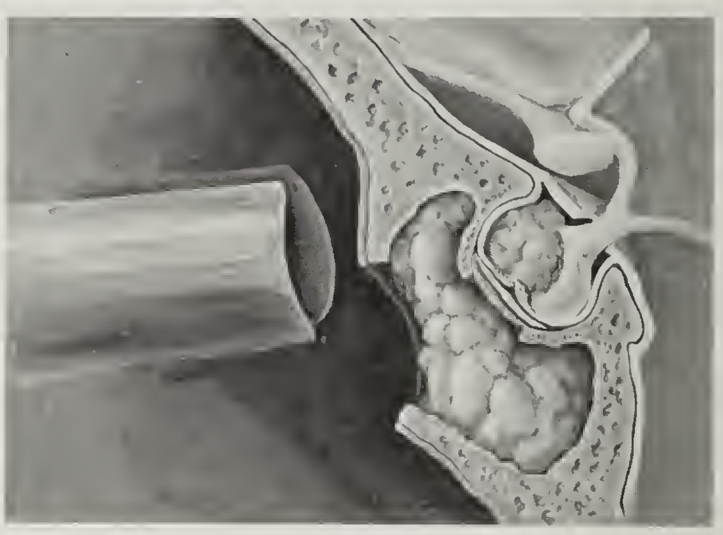

Figure 8
Reconstruction of sellar floor.

septum is removed and saved for reconstruction of the sellar floor. The entire piriform space is available to accept the self-retaining pituitary speculum.

The operating microscope is positioned in the surgical field. Under magnification, the anterior wall of the sphenoid sinus is removed and the sinus is exenerated (Figure 5). The pituitary speculum is carefully advanced into the sinus bringing the surgeon into a direct midline view of the floor of the sella turcica.

The floor is frequently thin and can easily be entered with a blunt instrument exposing the dura (Figure 6). If the floor is absent, the surgeon may encounter tumor directly under the mucosa of the sphenoid sinus. Following opening of the dura under high magnification, the distinction between tumor and normal gland is quite definite allowing removal of the former with preservation of the latter (Figure 7).

For hypophysectomy, the pituitary stalk is initially divided near the gland. Extracapsular dissection delivers the intact specimen into the sphenoid sinus for removal. Following completion of tumor removal or hypophysectomy, the emptied sella is loosely filled with muscle or adipose tissue obtained from the thigh. The previously removed piece of septum is shaped to form a new sellar floor and is held securely in place with a biological adhesive (Figure 8).

The nasal closure is rapid. Significant mucosal tears are repaired and the remaining septum is returned to a midline position. The gingival incision is closed with absorbable sutures. Placement of nasopharyngeal tubes and Gelfoam packing complete the operation.

Postoperative recovery is rapid and smooth. Discomfort is minimal and patients are taking oral nourishment within a few hours. Ambulation is started on the first p.o. day, nasal tubes and packing removed on the third p.o. day and discharge by the fifth to the seventh p.o. day.

Results

Our experience with a modest series of varied pituitary lesions over the past 24 months accurately reflects the larger series reported in the literature.[14, 15] Nineteen cases have been operated with no mortality.

Table 1
Indications

Sellar, suprasellar and infrasellar neoplasms.
Prolactin secreting microadenomas (amenorrhea, galactorrhea syndrome).
Total hypophysectomy for metastatic breast and prostatic cancer.
Intractable pain syndrome secondary to advanced neoplasia.
Sphenoid sinus lesions.
Post traumatic CSF rhinorrhea.

Table 2
Contraindications

1. Massive midline or lateral suprasellar tumor extension.
2. Suprasellar neoplasm with a normal sella.

Two of the earlier cases developed spinal fluid rhinorrhea; one of these progressed to meningitis, responsive to high level antibiotic therapy. The other case required reoperation to repack the sella and sphenoid sinus. The first patient experienced an unexplained unilateral central scotoma. Eight patients received postoperative irradiation. There has been no evidence of tumor recurrence to date. All nineteen patients resumed normal activity.

Discussion

General indications for transsphenoidal surgery are listed in Table 1. Of particular interest is the prolactin-secreting microadenoma responsible for the amenorrhea-galactorrhea syndrome in young women. the previously mentioned advances in endocrinology and radiology permit early diagnosis with increasing frequency. Selective removal of the microadenoma will allow resumption of a normal menstrual pattern with subsequent pregnancy in most instances. Excessive growth hormone produces acromegaly in the adult and gigantism in the child. Excessive ACTH with secondary cortisol elevation will lead to Cushing's disease. A variant of the latter is Nelson's syndrome occurring in approximately 10% of patients following bilateral adrenalectomy for Cushing's syndrome. All of these conditions can be successfully managed by this procedure.

Specific contraindications to this approach are outlined in Table 2. They relate directly to tumor size and disposition and sellar configuration. Massive tumor extension to the Foramen of Monro level or lateral extension into the middle cranial fossa cannot safely be managed by this midline route. Likewise a suprasellar mass with a normal sella is best approached from above. A poorly pneumatized or non-pneumatized sphenoid sinus (concha type) represents a relative contraindication.

The advantages of this surgical approach are several. Esthetically the procedure is more acceptable to the patient than craniotomy. Clipping and shaving of the hair is not necessary. There are no external scars since all incisions are submucosal. The frontal lobes are not retracted thereby obviating the possibility of injury to them. There is no loss of olfaction. Visual complications are lessened as there is reduced opportunity for manipulation.of the optic nerves and chiasm. Transient diabetes insipidus will occur in most patients, requiring several injections of Pitressin postoperatively. Hypophysectomy patients may require longer term intranasal vasopressin. The incidence of spinal fluid rhinorrhea has become an uncommon occurrence with water-tight reconstruciton of the sellar floor. This has, in addition, greatly reduced any likelihood of meningitis. Cosmetic defects of the nose, especially saddling, are infrequent now that the distal portion of the nasal septum is preserved.

Summary

Transsphenoidal surgery is the treatment of choice for most pituitary tumors. The approach allows successful removal of these lesions with preservation of normal pituitary function in a gratifying number of patients.

Acknowledgements

The author wishes to thank his associates, Drs. J.A. Eisenbeiss, W.B. Helme, and B.A. Kriegsfeld for the privilege of treating their patients included in this paper. Additionally, the assistance of Dr. Max Wertz in the development of the nasal portion of this procedure is much appreciated.

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The Human Agenda, Chapter 1 Prenatal Genetic Diagnosis

Harold D. Gabel, M.D.
Marilyn Laughead, M.D.

Using as tools the basic laws of heredity (genetics), and some sophisticated new techniques recently developed, the chance of bearing a child with a birth defect can often be predicted during the prenatal period. The state of Arizona now has a statewide Prenatal Genetic Diagnosis Program to provide just such a service.

The Arizona Perinatal Program with the co-sponsorship of the Arizona Department of Health Services and the Maternal and Child Health Care Committee of the Arizona Medical Association, has established this service. Arizona has already become a leader in the provision of high-quality perinatal services through such programs as the Newborn Transport Program and the Arizona Perinatal Program. This Prenatal Genetic Detection Program is yet another service for high-risk patients offered to the health care providers. It is based on the model for the care of other high-risk patients already established through the Arizona Perinatal Program. A health care provider may call the genetic-tertiary perinatal center in either Phoenix or Tucson to receive consultation and services for his or her maternity patient who may be genetically at risk.

Although specific birth defects may seem relatively uncommon, together they occur in about 7% of all births. The total number of affected families in the United States is well into the millions. Each year an additional 220,000 American babies are born with physical or mental defects of varying severity. Some of these defects, though present at birth, do not become apparent until months or years later.

There are approximately 43,000 births in Arizona each year. Of this group 2,000 are to women over the age of 35. This is one group of women who are at risk for bearing children with congenital anomalies. When other risk factors are considered, there are approximately 2,500 patients at risk each year in Arizona. The most prevaler abnormal genetic conditions that can be diagnosed ar Down's syndrome (Mongolism) and neural tube (spina cord) defects. Down's syndrome may occur at any tim in reproductive life but occurs in about one in every 10 pregnant women between the ages of 35 and 4 However, it occurs in 1 in 40 pregnancies over the age o 45. So it can be seen that the largest group at risk, in an pregnant population, are those women who ar pregnant and over the age of 35. Children affected wit neural tube defects occur in approximately 1 in 40 pregnant women. With a previously affected child th chance of another abnormal child increases t approximately 5 out of 100. The amniotic fluid alpha feto protein determination can detect this abnormality Eighty other metabolic abnormalities are als diagnosable prenatally by amniocentesis. Women wh may be at risk and should be considered eligible fo counseling about the Prenatal Genetic Diagnosi Program are:

1) Any woman 35 years of age or older at the time o conception;
2) Any woman with a child with Down's syndrome o another chromosomal abnormality;
3) Any woman carrying a chromosome abnormalit (or whose husband is carrying a chromosom abnormality);
4) Any woman with three or more consecutiv miscarriages;
5) Any woman with a family history of neural tub defect (such as anencephaly, spina bifida o meningomyelocele);
6) Any woman known to be a high-risk for a chil with recessive genetic disease which can be diagnose prenatally;
7) Any woman with a family history of severe X linked disease (which affects only boys);
8) Any woman with a child with multiple birth defect (of unknown cause).

For each woman at risk an found to be in need of services, a complete package o services is made available. This includes:

1) Genetic counseling related to the disease;
2) Amniocentesis with ultrasonic localization a recognized tertiary centers in Phoenix and Tucson;
3) Correct handling of specimens;
4) Laboratory studies including alpha-feto protein chromosome analysis with banding; and metaboli enzyme determination;
5) Follow-up.

Any pregnant woman may be served in Arizona b contacting her physician or health provider or she ma be assessed to be at risk by the physician or health provider. Alternatively, she may be assessed to be at risk by the physician or health provider during a prenatal care visit immediately or after her physician talks with a trained genetic counsellor in Phoenix or Tucson. Consultation and services may be obtained by calling in Phoenix, 894-1104, or in Tucson, 626-6324/626-6636. In

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From: Arizona Perinatal Program, 810 W. Bethany Home Road, Phoenix, AZ 85013.

some cases the need for prenatal diagnosis may be eliminated by a phone call between the referring physician and the genetic center. Eligible patients are then referred to a center in Phoenix or Tucson. The Arizona Perinatal Program's perinatal coordinators who already are functioning to deal with other high-risk conditions in pregnancies are available at the tertiary centers with other highly skilled professionals to provide the required genetic services. The referral center would be the choice of the referring physician.

When the patient arrives at the center, but prior to the amniocentesis for genetic studies, a visit with a genetic counselor is necessary. Whether the tests will be helpful might be determined by information provided during this interview. The Arizona Perinatal Program's perinatal coordinator would then perform the initial diagnostic amniocentesis required. The material obtained would then be tested in one of two designated laboratories: one in Phoenix and one in Tucson. These laboratories have been evaluated by the representative from the United States Center for Disease Control using recognized standards for genetic laboratories throughout the country. The total cost of the procedure would be approximately $400. This would include the entire package of the services mentioned previously except for transportation which the patient must provide. Payment sources for this procedure are usually the patient's own funds (a sliding scale has been employed for those who might need it) or third party carriers in some cases. The entire procedure can be performed in one visit. In some cases it is preferred that counseling and the amniocentesis be done on separate days.

The genetic counseling visit should be completed as early as possible. That is as soon as the pregnancy is confirmed. Amniocentesis is ideally performed between the 14th and 16th weeks of pregnancy, counted as the number of weeks after the beginning date of the last normal menstrual period. Earlier than the 13th week there is usually an insufficient amount of amniotic fluid to perform the test.

It is believed that amniocentesis is a relatively safe procedure. However, there are risks including the possibility that a miscarriage might occur afterwards. Currently, experienced centers have shown that the risks are low enough to warrant its use for the indications as listed above.

Techniques for prenatal diagnosis are available for only a specific number of disorders, there are many major diseases affecting growth and development of children which cannot be determined before birth, therefore, this test does not guarantee a normal baby in all patients. Because prenatal diagnosis is carried out in selected high-risk pregnancies and is a relatively new procedure, the reporting after all deliveries of any complications that might occur is essential.

An average of fourteen to twenty-one days is required for the report on chromosomes, a longer time is often necessary if the diagnosis involves biochemical determinations. Sometimes cell cultures must be sent to other laboratories in the United States for special tests. Occasionally cells in the original sample fail to grow or may grow very slowly, then the patient is requested to return for a second sample. If an abnormality is found, that information will be conveyed to the patient together with an assessment of the nature and degree of the probable impairment. The perspective parents can then make a choice as to the course of action they may wish to pursue. Follow-up subsequent to genetic studies is all part of the program and will be conducted from the genetic center from which the patient was served.

An educational program initially for physicians in Arizona about the need for and availability of prenatal diagnostic genetic services will be undertaken by the Arizona Perinatal Program in cooperation with other interested health professionals. This program will include brochures for physician's offices, for patient distribution, establishment of a "hot-line" for consultation services along with the Arizona Perinatal Program's high-risk consultation "hot-line" already in existence, regional continuing education conferences to include genetic high-risk information and articles in professional health care journals. All efforts will be directed towards using "approved" centers which offer a complete package of services at the highest quality. The genetic centers will also be open for physician's visits to augment their own continuing education programs in genetics.

A public education program will also be inaugurated to insure that regional genetic centers in Tucson and Phoenix are known to the general population. This will be done through the use of radio, television, newspapers, and through the use of presentations to groups such as P.T.A.'s and Women's Clubs. Posters and informational brochures will also be available for statewide distribution.

Abnormal findings are rare, in fact they occur in about two percent of all cases. Almost all of those patients who have taken the tests will be able to relax for the rest of their pregnancy and be assured regarding the risks from a genetic point of view.

In today's legal climate and because it is a part of high-quality obstetric care, it is incumbent upon all health care providers to screen their prenatal patients whether seen in clinics or in the private office for those conditions which will place them at risk for bearing a genetically defective infant. Having identified such a patient the availability of prenatal genetic detection services augment the armamentarium of the practicing physician and make him even more well equipped to deal with all conditions that present obstacles to achieving the best possible outcome of pregnancy for his or her patient and her unborn child.

Genetic services may be obtained by calling in Phoenix, 894-1104, or in Tucson, 626-6324/626-6636.

Oncology

Lithium Carbonate Stimulation of Granulopoiesis in Hematologic and Oncologic Disease

Thomas Anderson, M.D.

Editor:
David Alberts, M.D.

Introduction

For years lithium therapy has been the treatment of choice for manic-depressive disorders. A curious side effect of lithium treatment in psychiatric and non-psychiatric patients has been granulocytosis.[1] At normal psychiatric doses (900-1200 mg per day) the granulocytosis occurs within 1-2 weeks and persists as long as the drug is given. Lithium therapy is associated with an increase in a blood factor known as colony-stimulating activity (CSA), which induces the bone marrow to increase neutrophil production.[2] Measurement of circulating granulocytes, granulocyte marrow reserve, and marginated granulocytes shows that there is a true increase in granulocyte production rather than a redistribution phenomenon.[3]

Clinical Pharmacology of Lithium

Lithium in the carbonate salt form is 99 percent absorbed after an oral dose and peak blood levels are reached in 1-3 hours, with absorption usually complete by 8 hours. Once in the blood, lithium is not bound to protein and is distributed into various tissues at non-uniform rates. The average plasma half-life is about 24 hours, varying from 18 hours in younger persons to 36 hours in older persons. Elimination is dependent upon renal excretion and this, in turn, depends on sodium balance. Salt-free diets, diuretics, extensive exercise, and diarrhea can all result in lithium retention.

With the therapeutic doses used in manic-depressive disorders, the serum concentrations of lithium range between 0.6 and 1.2 meq/liter. The most common side effects of initial lithium treatment include gastrointestinal upset, a fine hand tremor, fatigue drowsiness, and sometimes polyuria. Lithium intoxication is usually manifested as an unsteady gait muscle twitching, and slurred speech. This can usually be managed by discontinuing the lithium. Severe intoxication can lead to coma and possible death.

Lithium Usage in Non-malignant Disease

The clinical implications of lithium-induced leukocytosis have received considerable attention in the recent medical literature. Gupta and Robinson[4] reported that patients with Felty's syndrome (rheumatoid arthritis, neutropenia, and splenomegaly) have increases in their blood neutrophil concentrations when taking lithium. The numbers of patients were small however, and it is not clear whether or not these patients had fewer infections.

Lithium has also been used with mixed success in the management of aplastic anemia[5] and congenital neutropenia.[6] In the two treated patients with aplastic anemia, the neutrophil counts doubled and were maintained for 5+ months in one patient, but for only one month in the other patient. One of two pediatric patients with congenital neutropenia responded with a brief normalization of neutrophil counts and clearing of a chronic infection, while in the other patient, no beneficial effect was demonstrated.

Lithium Usage in Malignant Disease

The most recent and potentially important use of lithium has been in patients with cancer. The treatment of malignant disease is often limited by hematologic toxicity from anticancer drugs, radiation therapy, or both. A reduction of neutropenia might decrease episodes of infection and, in turn, this may allow increased drug dosage, and theoretically, increased tumor cell kill. With these goals in mind, several investigators have initiated lithium studies in patients with cancer.

Greco[7] studied patients with advanced lymphomas and bronchogenic carcinoma. In the 16 evaluable patients receiving consecutive chemotherapy courses without and with lithium, the mean neutrophil nadir count during the lithium treatment was 26% higher than that in the pre-lithium cycle and 40% higher than that in the post-lithium cycle. In a similar study, Stein[8] reported that the mean neutrophil nadir count and neutrophil count at the end of the chemotherapy cycle of 13 patients with cancer were significantly ($p<0.05$) higher during the chemotherapy cycles with lithium.

Most recently, Stein[9] has reported granulocyte data on 27 patients receiving standard intensive chemotherapy for acute myelogenous leukemia and randomized to receive lithium carbonate (300 mg TID) or no lithium carbonate. In comparable treatment

From: Section of Hematology and Oncology, Dept. of Internal Medicine, Univ. of Arizona Health Sciences Center, 1501 North Campbell Ave., Tucson, AZ 85724

groups, patients had granulocyte nadirs below 100/mm³; however, the median duration of granulocytopenia (<1000/mm³) was 16.0 days in the lithium-treated group and 24.6 days in the group not receiving lithium ($p<0.013$). Despite the shortened duration of neutropenia, however, neither the incidence of infection nor the rate of remission was affected. Lyman[10] has reported on 35 patients with oat cell carcinoma of the lung who were intensively treated with a combination of chemotherapy and radiation and who were randomly assigned to lithium or no lithium. Treatment with lithium significantly increased the platelet count and granulocyte count, and decreased both the need for chemotherapy dosage reduction and the incidence of clinical infection. During the trial, four infection-related deaths occurred (all in the control [no lithium] group). Other investigators have substantiated the beneficial effect on the platelet count[11] and in the prevention of chemotherapy-related infections.[12]

The University of Arizona Cancer Center will soon initiate a randomized, prospective clinical trial of lithium carbonate therapy during antitumor chemotherapy. Although most other trials have used standard psychiatric maintenance doses (900 mg/day) of lithium, there is little reason to correlate psychiatric benefit with granulocytosis. Moreover, since several patients at the University of Arizona have had increased granulocyte counts on a once-a-day, low dose of lithium, and since lithium toxicity has been reported in previous trials using higher doses, we will begin with a low-dose, double-blind evaluation of lithium stimulation of granulopoiesis during cancer chemotherapy. We hope that these studies will lead to a simple, non-toxic method for preventing the need for reduction in antitumor drug dose and will decrease the incidence of chemotherapy-related infection.

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Special Article

The Compleat Physician*

Max Costin, M.D.

Introduction

As editor I have the "honor" of reviewing all manuscripts before they are sent for independent perusal. It is rare that I find an unsolicited manuscript which needs no such outside critical observation. The article which follows is an example of such a "find". It needs to be carefully read by all of us who consider ourselves physicians.
MBB

Synopsis-Abstract

A subjective assessment, derived mainly from patient interviews is presented in an effort to construct a model of the "good doctor" from the standpoint of the patient. The essential elements are summarized and discussed under four rubrics (The Four C's):

Competence
Concern
Credibility
Communicative Skills

It is concluded that certain "know-hows" could be mastered which would make the model of the 'Compleat Physician' an approachable reality.

From: Dept. of Obstetrics and Gynecology, University of Arizona Health Sciences Center, Tucson, AZ 85724 (Dr. Costin, Clinical Associate and Consulting Gynecologist, Student Health Service.

*Paraphrase of "The Compleat Angler" by Isaak Walton -1653.

The Four C's

A funny thing happened to me on my way to the completion of my tenth year of medical practice. Through the patient-friend grapevine I learned that, in the opinion of some of my patients, I lacked a few of the qualities I needed to become a "real good doctor." I thought about that for some time and asked questions of my patients and peers, and soon realized that I had much to learn about what constituted a "good doctor" in the eyes of the patient.

For the two decades since that time, I have been listening to what my patients have been saying concerning the ingredients which they believe are needed in the synthesis of the "Compleat Physician". In attempting an analysis of these ingredients, I have telescoped them under four rubrics: *competence* (knowledge, expertise, know-how); *concern* (caring, empathy, compassion, kindness); *credibility* (trustworthiness, promise-keeping, commitment, self-imposed responsibility, reliability); *communicative skills* (understanding, ability to inform clearly, ability to listen). There follow, within the framework of these four categories, comments distilled from patient encounters and the humanistic literature - tinctured with my own bias.

Competence

"Reason's whole pleasure, all the joys of sense,
Lie in three words, health, peace and competence."

Alexander Pope

Almost everyone would agree that competence is the sine qua non of an effective physician. The problem seems to be that the physician and his patient may define competence from somewhat different viewpoints. Most doctors perceive it in the context of scientific knowledge, clarity of reasoning, and perhaps manual dexterity. The patient requires that the doctor's expert technology be person oriented. Knowledge and expertise are certainly humane when they are used in the service of the trust that the patient places in the doctor's ability to help and not to harm (Primum non nocere). Unfortunately a physician may abuse his competence. He may become so engulfed by his highly technical ambience, or so entranced by a procedure in which he has gained much proficiency, that his own diagnostic or therapeutic goose becomes transformed into a swan. To him the patient may become a mere "container" of a pathological process which needs to be identified and conquered. Since, "when your only tool is a hammer, every problem becomes a nail." (O.W. Holmes), he may tend to recommend and perform his favored procedure even when benefits are marginal or totally absent. It is at this point that competence becomes inappropriate and inhumane.

There is no doubt that new biological theories, important scientific conjectures, and even new "facts" are flourishing, and that highly sophisticated technology now plays an increasing role in the practice of medicine. The physician must be knowledgeable about these advances. However, in that consortium of clinicians, teachers, researchers, and machines needed to carry on high quality medical care today, the machine must not be allowed to become the senior partner, lest society end up with a dehumanizing mechanization of medicine.

There is an important benefit of competency which is of unestimable value to the physician and patient alike. The deeply satisfying awareness of one's own depth of knowledge and competency promotes feelings of self-worth and confidence which, in turn, translate to enhancement of positive therapeutic results, a distinct patient benefit.

Concern

"Experts understand and deal well with diseases;
Physicians should understand and deal well with people;
Oh that all of us could be physician experts."

William Barclay, M.D.

Competence without concern makes a good hangman. Knowledge and compassion are not mutually exclusive. Our "good doctor" will show concern for his patient; not exhibited as anxiety nor perfunctory caring, but in the form of controlled empathic interest, with sensitivity to the attitudes, values, and feelings of the patient—a concern for the whole patient rather than for the patient's disease alone.

Some patients complain that their physicians exhibit a degree of concern and caring that is far below the level of their congnitive skills. While these doctors can presumably identify in others characteristics such as concern, empathy, and compassion, they find themselves unable to express these elements in their patient relationships. It is easier to convince oneself that one loves mankind (abstract), than it is to enter into a doctor-patient relationship displaying genuine respect and compassion toward the patient (concrete). To paraphrase a Chinese philosopher, it is very easy to draw a goblin, but very difficult to draw a horse.

Much has been written on the topic of caring for the patient. Michael Balint writes, "The most effective drug the doctor furnishes to his patient is the doctor himself. There is no pharmacopeia that will tell you the starting or the maintenançe dose, or how long it has to be administered."[1] In his oft-quoted paper of 1927, Peabody states, "The good physician knows his patient through and through, and his knowledge is bought dearly. Time, sympathy, an understanding must be lavishl dispensed, but the reward is to be foun in the personal bond which forms th greatest satisfaction of the practice o medicine. One of the essential qualities o the clinician is interest in humanity fo the secret of the care of the patient is i the caring for the patient."

From the patient's standpoint, th experience of seeking and receivin medical aid involves a good deal of stres and anxiety as well as feelings o inferiority, resentment and even shame To meet the patient's needs, the physicia must deal with these feelings. In additio to addressing himself to a specific orga dysfunction, the "good doctor" can b expected to attend to the "dis-ease" of hi patient by allaying anxieties an supporting or increasing the patients' feelings of self-worth.

The physician's caring investment i the patient is not free of side effects Although coincidentally effective as therapeutic tool, the caring and empathi approach exacts from the doctor considerable emotional and physical toll As the physician becomes deeply involve with troubled human beings and thei biological, psychological, and socia problems, he may at some point lose hi capacity to cope with the emotional strai engendered. As a result he may begin t exhibit the symptoms of the Burned-ou Syndrome.[3] In this state, the docto withdraws, fails to display empathy toward his patients, and often begins t deal with them in a detached manner This detachment phenomenon may take the form of physical distancing an hurrying through of patients, and even an irascible use of denigrating language (what a crock!). The physician may dor such a thick armor that nobody can ge through. An early self-recognition of this syndrome is essential, so that, after self-evaluation, readjustments can promptly be carried out. The dysfunctiona behavior can then be self-limiting, and a previously sensitive and caring physician can heal himself.

Credibility

"The mutual confidence on which all else depends can be maintained only by an open mind and a brave reliance upon free discussion."

Learned Hand

The most frequently used word that I heard from my patients when referrals were being made was **trust**: "I want a doctor I can trust." In retrospect, it is obvious that credibility rated a high priority. I believe that it also looms large to most physicians as a desirable professional quality. The importance of trustworthiness in this context is admirably summarized by Robert E. Cooke:

"If one takes a formalist approach to the series of transactions involved in medical care, trust appears to be a critical agreement. Trust is the consequence of several basic ethical principles including promise-keeping, beneficence, non-injury, and justice. Without trust the relationships between physician and patient cannot rise above that between provider and consumer. In certain respects this difference may be the difference between humanized and dehumanized care. If the physician is regarded only as a provider when problems so important as life and death are concerned, then medical care is dehumanized."[4]

Trustworthiness also implies responsibility and courage to commit oneself completely to the action. This is not an easy accomplishment in our times. C.D. Aring writes, "We live in an era where major political events and economic and social problems contribute to the devaluation of honesty and mercy, dignity and love, and respect and fair play."[5]

Trust can promote healing whether it is the context of treating a bodily dysfunction or counseling a relationship problem. In the words of Norman Cousins, "A doctor who enjoys the full trust of his patients can activate the body's benevolent chemistry in the same way as does a placebo. Such a doctor has a therapeutic effect on his patients just in the act of removing their anxieties and giving them confidence in the recuperative capactiy of their own bodies."[6] The late Felix Marti-Ibanez believed, according to his writings, that through kindness, courtesy, and honesty, "you can give them (patients) relief often, but hope you can give them always."

Communicative Skills

"A word is dead
When it is said
Some say.
I say it just begins to live
That day."

Emily Dickinson

Most patients ostensibly want a doctor who can divine the nature of their illnesses and remedy them without too much investment on the part of the patient of time, effort, and money. But they want and need much more than that. Many we see in our offices are suffering not only from organic or functional disorders, but from maladies of the spirit as well. To maximally aid these patients, the physician needs to master some communicative skills. It is the deft employment of these communicative skills that is, I believe, the essence of the Art of Medicine.

The doctor-patient relationship involves a series of transactions. These messages can be constructive or destructive. One cannot **not** communicate. In a medical encounter, both the patient and the doctor are continually broadcasting (or telecasting) their attitudes and feelings, both verbally and nonverbally. The physician obviously needs to avoid destructive communication which may engender **iatrogenic illness** in the patient. In my opinion, it is even more essential that he should utilize constructive communication to encourage **iatrogenic health.** Positive iatrogenesis should be a part of every doctor's armamenatium. Constructive communication is non-judgemental. If one becomes judgemental of another, one creates a barrier to any effective therapeutic relationship.

A neglected facet of constructive communication is the art of listening. The next time you are interviewing a patient, make a conscious effort to "listen to your listening." Were you really aware of the true message? Were you conscious of the non-verbal cues as well as the words spoken; the patient's attitudes, body movements, posture, flushing and other nuances of the patient's behaviour? If not, did you receive all the message sent? As a busy physician with a tight schedule pushing you, have you developed a tympanic barrier? If I were arbitrarily limited to developing skill in only one facet of constructive communication with patients, I would select listening skill. Many times it is the only therapy needed.

The word doctor is derived from the Latin "docere", to teach. Most patients today want to make more decisions regarding their health, or at least take a more active role in the decision-making process. To do this they need more information than they are ordinarily given. Herewith lies a communicative gap which often exists between doctor and patient. Reducing anxiety while at the same time closing this gap offers a real challenge. It is certainly appropriate for the doctor, as teacher, to create an ambience conducive to learning (educare, L. - to lead out). One goal of patient eduction should be to seek out and dispel the misconceptions about their health problems that so many harbor. Another aim is to offer the patient information in an understandable and non-threatening manner. In the process of informing a patient, it is essential that what you tell him or her be completely true, though a certain portion of the entire truth may be omitted at the discretion of the physician should he feel it would be destructive to the well-being of the patient at that point in time.

Summary

Most patients, upon being referred, will request transfer to a good doctor. Patients' notions about what qualifies a physician to receive this approbation differ, sometimes widely, from the judgements of the physician's colleagues. The attributes most frequently mentioned by patients as drawing their approval and respect have been outlined and briefly discussed.

It should be clear that our "good doctor" is neither a reality nor a fantasy. She/he is a model. Does the Compleat Physician belong to a vanishing era? I think not. Attaining perfection in the standards of the four C's is probably an impossibility, but I believe that there are certain "know-hows" which, if mastered, could make the goal of the Compleat Physician approachable. Here are the messages which I think my patients, pertinent literature, and my own conscience have been sending me:

Know the extent of your knowledge and expertise and maintain the motivation (curiosity?) to expand them.

Know how to display disciplined compassion.

Know how to show consideration and respect toward others.

Know how to communicate constructively and nonjudgmentally.

Know how to think like a scientist and behave like a humanist.

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State of the Art

The Pathogenesis of Diabetes Mellitus

David G. Johnson, M.D.

Editor:
Eric H. Prosnitz, M.D.

Recent advances in our knowledge of diabetes mellitus have drawn attention to the multiple causes and pathophysiologic alterations that can lead to this disorder. Clinically the term "diabetes mellitus" refers to any disease process characterized by hyperglycemia and glycosuria. If this abnormality in glucose metabolism is clearly the result of a more definable lesion, such as pancreatitis, Cushing's syndrome, or hemachromatosis, it is common to refer to the diabetes as "secondary" to the underlying disease process. However, the vast majority of patients with diabetes mellitus do not have such identifiable causes for their disease. Study in many areas of research, including genetics, virology, immunology, and biochemistry has begun to suggest several pathogenetic processes that may be responsible for the "primary" forms of diabetes.

Regardless of the type or cause of diabetes mellitus, most of the metabolic abnormalities are, at least in part, the result of a deficiency in the actions of insulin on its target tissues. The lack of insulin-controlled effects in the body can be due to either inadequate secretion of insulin from the B-cells of the pancreas or an inadequate response to insulin in the major target tissues—liver, muscle, and adipose tissue. Both mechanisms - impaired secretion of insulin and defective tissue responses - are present in many patients. Several pathologic processes can occur in either the pancreatic islet or peripheral tissues to interrupt the normal hormonal influence of insulin on metabolism.

From: Univ. of Arizona Health Sciences Center, Sect. of Endocrinology, Tucson, AZ 85724

Tenuate® ©IV
(diethylpropion hydrochloride NF)
Tenuate Dospan®
(diethylpropion hydrochloride NF) controlled-release

AVAILABLE ONLY ON PRESCRIPTION
Brief Summary

INDICATION: Tenuate and Tenuate Dospan are indicated in the management of exogenous obesity as a short-term adjunct (a few weeks) in a regimen of weight reduction based on caloric restriction The limited usefulness of agents of this class should be measured against possible risk factors inherent in their use such as those described below

CONTRAINDICATIONS: Advanced arteriosclerosis, hyperthyroidism, known hypersensitivity, or idiosyncrasy to the sympathomimetic amines, glaucoma Agitated states Patients with a history of drug abuse. During or within 14 days following the administration of monoamine oxidase inhibitors, (hypertensive crises may result).

WARNINGS: If tolerance develops, the recommended dose should not be exceeded in an attempt to increase the effect; rather, the drug should be discontinued. Tenuate may impair the ability of the patient to engage in potentially hazardous activities such as operating machinery or driving a motor vehicle, the patient should therefore be cautioned accordingly *Drug Dependence* Tenuate has some chemical and pharmacologic similarities to the amphetamines and other related stimulant drugs that have been extensively abused. There have been reports of subjects becoming psychologically dependent on diethylpropion. The possibility of abuse should be kept in mind when evaluating the desirability of including a drug as part of a weight reduction program Abuse of amphetamines and related drugs may be associated with varying degrees of psychologic dependence and social dysfunction which, in the case of certain drugs, may be severe. There are reports of patients who have increased the dosage to many times that recommended Abrupt cessation following prolonged high dosage administration results in extreme fatigue and mental depression; changes are also noted on the sleep EEG. Manifestations of chronic intoxication with anorectic drugs include severe dermatoses, marked insomnia, irritability, hyperactivity, and personality changes. The most severe manifestation of chronic intoxications is psychosis, often clinically indistinguishable from schizophrenia *Use in Pregnancy*: Although rat and human reproductive studies have not indicated adverse effects, the use of Tenuate by women who are pregnant or may become pregnant requires that the potential benefits be weighed against the potential risks. *Use in Children*. Tenuate is not recommended for use in children under 12 years of age.

PRECAUTIONS: Caution is to be exercised in prescribing Tenuate for patients with hypertension or with symptomatic cardiovascular disease, including arrhythmias Tenuate should not be administered to patients with severe hypertension. Insulin requirements in diabetes mellitus may be altered in association with the use of Tenuate and the concomitant dietary regimen. Tenuate may decrease the hypotensive effect of guanethidine. The least amount feasible should be prescribed or dispensed at one time in order to minimize the possibility of overdosage. Reports suggest that Tenuate may increase convulsions in some epileptics. Therefore, epileptics receiving Tenuate should be carefully monitored. Titration of dose or discontinuance of Tenuate may be necessary

ADVERSE REACTIONS: *Cardiovascular*: Palpitation, tachycardia, elevation of blood pressure, precordial pain, arrhythmia. One published report described T-wave changes in the ECG of a healthy young male after ingestion of diethylpropion hydrochloride. *Central Nervous System*: Overstimulation, nervousness, restlessness, dizziness, jitteriness, insomnia, anxiety, euphoria, depression, dysphoria, tremor, dyskinesia, mydriasis, drowsiness, malaise, headache, rarely psychotic episodes at recommended doses In a few epileptics an increase in convulsive episodes has been reported. *Gastrointestinal*: Dryness of the mouth, unpleasant taste, nausea, vomiting, abdominal discomfort, diarrhea, constipation, other gastrointestinal disturbances. *Allergic*: Urticaria, rash, ecchymosis, erythema, *Endocrine*: impotence, changes in libido, gynecomastia, menstrual upset. *Hematopoietic System*. Bone marrow depression, agranulocytosis, leukopenia *Miscellaneous* A variety of miscellaneous adverse reactions has been reported by physicians. These include complaints such as dyspnea, hair loss, muscle pain, dysuria, increased sweating, and polyuria.

DOSAGE AND ADMINISTRATION: Tenuate (diethylpropion hydrochloride): One 25 mg. tablet three times daily, one hour before meals, and in midevening if desired to overcome night hunger Tenuate Dospan (diethylpropion hydrochloride) controlled-release: One 75 mg tablet daily, swallowed whole, in midmorning. Tenuate is not recommended for use in children under 12 years of age.

OVERDOSAGE: Manifestations of acute overdosage include restlessness, tremor, hyperreflexia, rapid respiration, confusion, assaultiveness, hallucinations, panic states. Fatigue and depression usually follow the central stimulation. Cardiovascular effects include arrhythmias, hypertension or hypotension and circulatory collapse. Gastrointestinal symptoms include nausea, vomiting, diarrhea, and abdominal cramps. Overdose of pharmacologically similar compounds has resulted in fatal poisoning, usually terminating in convulsions and coma. Management of acute Tenuate intoxication is largely symptomatic and includes lavage and sedation with a barbiturate. Experience with hemodialysis or peritoneal dialysis is inadequate to permit recommendation in this regard Intravenous phentolamine (Regitine®) has been suggested on pharmacologic grounds for possible acute, severe hypertension, if this complicates Tenuate overdosage.

Product Information as of April, 1976

MERRELL-NATIONAL LABORATORIES Inc
Cayey, Puerto Rico 00633

Direct Medical Inquiries to
MERRELL-NATIONAL LABORATORIES
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References: 1. Citations available on request from Medical Research Department, MERRELL-NATIONAL LABORATORIES, Cincinnati, Ohio 45215 2. Hoekenga, M T., O'Dillon [Dillon], R H., and Leyland, H M. A comprehensive review of diethylpropion hydrochloride. In, Central Mechanisms of Anorectic Drugs, S Garattini and R. Samanin, Ed., New York, Raven Press, 1978, pp 391-404

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9-4672 [Y957A]

Overweight may not always be simple... complications can develop.* Complicated or not...

Tenuate® Dospan® ⒸIV

(diethylpropion hydrochloride NF)

75 mg. controlled-release tablets

A useful short-term adjunct in an indicated weight loss program.

Overweight patients in certain diagnostic categories often require strict appetite control and a successful program of weight reduction may tend to diminish the incidence or severity of the complications in some patients. Diethylpropion hydrochloride has been reported useful in such patients and while it is not suggested that Tenuate itself in any way reduces the complications of overweight, it may have a useful place as a short-term adjunct in a prescribed dietary regimen. **Tenuate should not be administered to patients with severe hypertension; see additional Warnings and Precautions on the opposite page.**

In uncomplicated overweight.

Many patients, on the other hand, present with excess fat but no disease. While this condition is often termed uncomplicated obesity, complications of both a social and a psychologic nature may be distressingly real for the patients. In these cases, a short-term regimen of Tenuate can help reinforce your dietary counsel during the important early weeks of an indicated weight loss program.

Clinical effectiveness.

The anorectic effectiveness of diethylpropion hydrochloride is well documented. No less than 16 separate double-blind, placebo-controlled studies attest to its usefulness in daily practice.[1] And the unique chemistry of Tenuate provides "...anorectic potency with minimal overt central nervous system or cardiovascular stimulation."[2] Compared with the amphetamines, diethylpropion has minimal potential for abuse.

Tenuate–it makes sense. And it's responsible medicine.

*Studies have shown that obesity is associated with an increased incidence of hypertension, symptomatic heart disease, adult-onset diabetes, and other diseases.

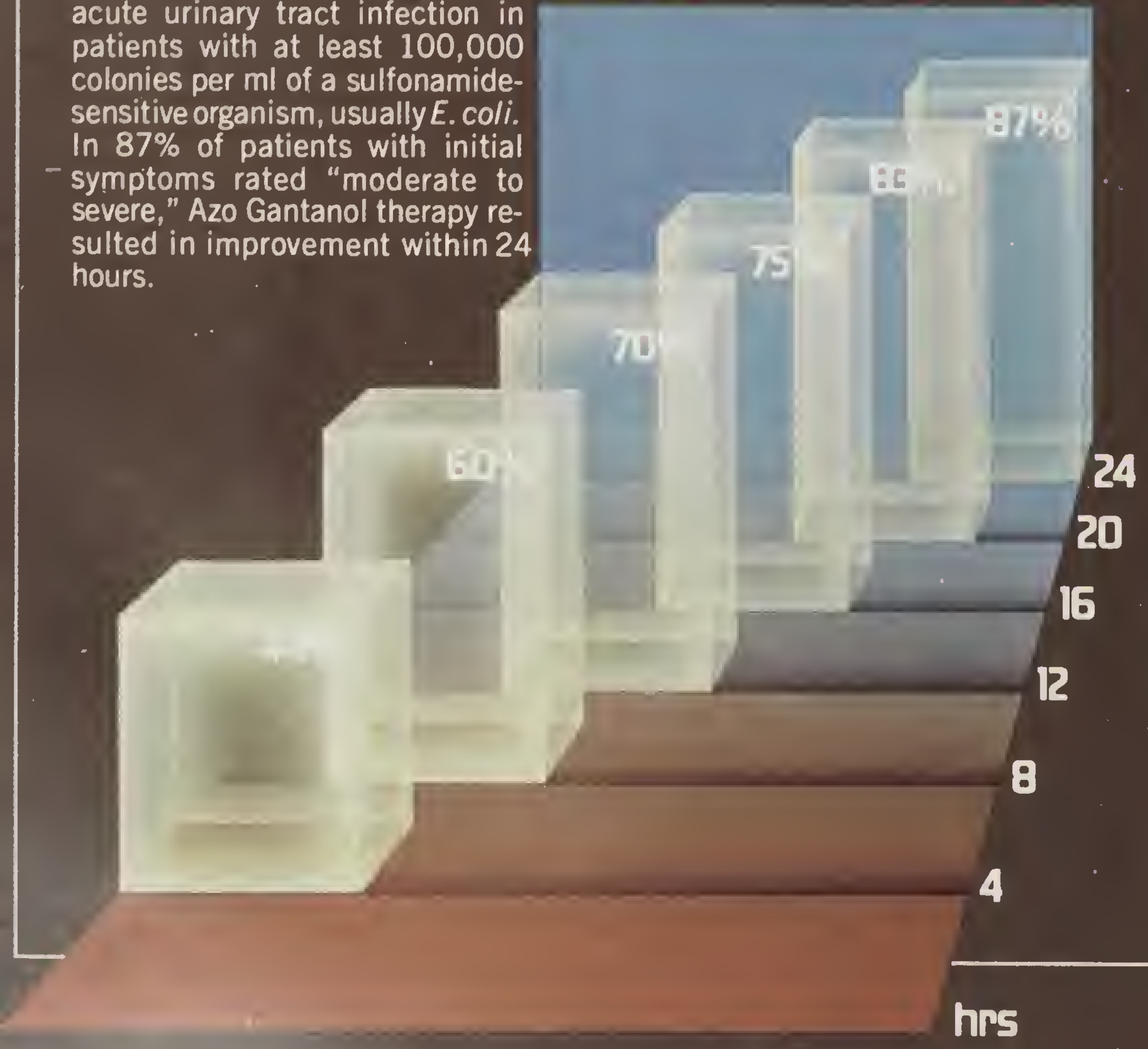
Merrell

For prescribing information see opposite page

Important data on the pain of acute cystitis:

In 87% of patients studied [303 of 349], Azo Gantanol® reduced pain and/or burning within 24 hours*

A controlled, multicenter study assessed the efficacy of Azo Gantanol in relieving pain and/or burning associated with acute urinary tract infection in patients with at least 100,000 colonies per ml of a sulfonamide-sensitive organism, usually *E. coli*. In 87% of patients with initial symptoms rated "moderate to severe," Azo Gantanol therapy resulted in improvement within 24 hours.



Fast pain relief plus effective antibacterial action

Azo Gantanol®

Each tablet contains 0.5 Gm sulfamethoxazole and 100 mg phenazopyridine HCl.

for
the pain

for
the pathogens

*Data on file, Hoffmann-La Roche Inc., Nutley, New Jersey 07110.

Before prescribing, please consult complete
uct information, a summary of which follows:
Indications: In adults, urinary tract infection
complicated by pain (primarily pyelonephritis
pyelitis and cystitis) due to susceptible organ
(usually *E. coli, Klebsiella-Aerobacter, Staph*
coccus aureus, Proteus mirabilis, and, less fr
quently, *Proteus vulgaris*) in the absence of
obstructive uropathy or foreign bodies. **Note:**
fully coordinate *in vitro* sulfonamide sensitivi
tests with bacteriologic and clinical response
aminobenzoic acid to follow-up culture media
Increasing frequency of resistant organisms li
the usefulness of antibacterials including sul
fonamides. Measure sulfonamide blood levels
variations may occur; 20 mg/100 ml should b
maximum total level.
Contraindications: Children below age 12; su
fonamide hypersensitivity; pregnancy at term
during nursing period; because Azo Gantanol
tains phenazopyridine hydrochloride it is con
dicated in glomerulonephritis, severe hepatiti
uremia, and pyelonephritis of pregnancy with
disturbances.
Warnings: Safety during pregnancy not establ
Deaths from hypersensitivity reactions, agran
tosis, aplastic anemia and other blood dyscra
have been reported and early clinical signs (s
throat, fever, pallor, purpura or jaundice) may
dicate serious blood disorders. Frequent CBC
urinalysis with microscopic examination are r
ommended during sulfonamide therapy.
Precautions: Use cautiously in patients with i
paired renal or hepatic function, severe allerg
bronchial asthma; in glucose-6-phosphate
dehydrogenase-deficient individuals in whom
dose-related hemolysis may occur. Maintain
adequate fluid intake to prevent crystalluria a
stone formation.
Adverse Reactions: *Blood dyscrasias* (agran-
ulocytosis, aplastic anemia, thrombocytopeni
leukopenia, hemolytic anemia, purpura, hypo
thrombinemia and methemoglobinemia); *alle*
reactions (erythema multiforme, skin eruptio
Stevens-Johnson syndrome, epidermal necrol
urticaria, serum sickness, pruritus, exfoliative
dermatitis, anaphylactoid reactions, periorbit
edema, conjunctival and scleral injection, ph
sensitization, arthralgia and allergic myocard
G.I. reactions (nausea, emesis, abdominal pa
hepatitis, diarrhea, anorexia, pancreatitis and
stomatitis); *CNS reactions* (headache, periph
neuritis, mental depression, convulsions, atax
hallucinations, tinnitus, vertigo and insomnia
miscellaneous reactions (drug fever, chills, to
nephrosis with oliguria and anuria, periarterit
nodosa and L. E. phenomenon). Due to certa
chemical similarities with some goitrogens, d
uretics (acetazolamide, thiazides) and oral hy
glycemic agents, sulfonamides have caused r
instances of goiter production, diuresis and h
glycemia. Cross-sensitivity with these agents
exist.
Dosage: Azo Gantanol is intended for the acu
painful phase of urinary tract infections. *Usua*
adult dosage: 2 Gm (4 tabs) initially, then 1
(2 tabs) B.I.D. for up to 3 days. If pain persis
causes other than infection should be sought.
After relief of pain has been obtained, contin
treatment with Gantanol (sulfamethoxazole) n
be considered.
NOTE: Patients should be told that the orang
dye (phenazopyridine HCl) will color the urine
Supplied: Tablets, red, film-coated, each con
ing 0.5 Gm sulfamethoxazole and 100 mg
phenazopyridine HCl—bottles of 100 and 50

ROCHE

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Heterogeneity of Diabetes Mellitus

Ever since the studies of Himsworth in the 1930's it has been recognized that patients with diabetes mellitus can be separated into two, broad, groups, depending on their sensitivity to insulin.[1] Patients prone to ketosis are usually sensitive to insulin, whereas older, often obese, individuals with non-ketotic diabetes tend to be insensitive to the effects of insulin.[2] Using bioassay to measure plasma insulin activity, Bornstein and Trewhella reported in 1950 that diabetic patients with weight loss and ketosis had no detectable insulin activity, while obese, non-ketotic diabetics had essentially normal insulin activity.[3] Following the development of radioimmunoassay techniques to measure insulin in the early 1960's it became clear that most patients with adult-onset (ketosis-resistant) diabetes have considerable amounts of circulating insulin.[4] Obese individuals without diabetes have even higher plasma insulin concentrations.[5] This suggested that obesity induces a resistance of target tissues to the action of insulin. In patients with the adult onset (non-ketotic) kind of diabetes the resistance to insulin's effects is not compensated adequately by increased insulin secretion.[6]

Newly diagnosed, juvenile-onset diabetics have low plasma concentrations of insulin. The antibodies against insulin that develop subsequently in insulin-dependent (juvenile-onset) diabetes interfere with the radioimmunioassay of plasma insulin.

However, using a radioimmunoassay for C-peptide, a fragment of proinsulin that is secreted in equimolar amounts with insulin, it is possile to estimate the release of endogenous insulin.[7] After several years of disease, most patients with juvenile-onset diabetes have little or no endogenous secretion of insulin.[8]

Viral-induced Diabetes Mellitus

For many years there have been occasional reports of the acute onset of diabetes mellitus in association with viral infections. Recent studies have provided strong evidence for an etiologic role of certain viruses in the development of diabetes mellitus.[9] Epidemiologic data have drawn attention to the association of mumps, infectious mononucleosis, hepatitis, rubella and Coxsackie B4 infections and the onset of diabetes mellitus. Yoon et al[10] have isolated a varient of Coxsackie B4 virus from the pancreas of a patient with newly diagnosed diabetes. Histologic examination of the patient's pancreas revealed an acute inflammatory process in the pancreatic islets with lymphocytic infiltration. The virus also attacked mouse pancreatic B-cells cultured *in vitro*. This suggests that, in a susceptible host, viral infection can injure pancreatic islet cells, leading to impaired insulin secretion and diabetes mellitus.

Autoimmunity and Histocompatibility Typing

Immunologic studies indicate that many individuals with juvenile-onset diabetes have antibodies in their plasma that react with pancreatic islet cells.[11] These antibodies may mediate both homoral and cellular immune response that can injure or destroy panreatic B-cells. The observation that titers of anti-islet cell antibody are highest in the earlier stages of clinical disease suggests that an inciting agent, such as a virus, may damage the B-cell, leading to an autoimmune response against the B-cell that results in the later development of diabetes. Interestingly, a rodenticide that is toxic to pancreatic B-cells has also been show to induce formation of islet cell antibodies and lead to the development of diabetes mellitus.[12] It is possible that diabetes that occurs immediately during or after a viral infection or exposure to toxin is due to direct injury to the B-cells, whereas diabetes that develops a variable time interval later may be largely the result of autoimmune-mediated injury.

Additional evidence linking insulin-dependent diabetes mellitus to abnormal immunologic responses comes from a study of histocompatibility (HLA) genotypes.[13] Patients with insulin-dependent diabetes have a higher frequency of certain HLA genotypes, including HLA B8, B18 (Southern Europeans), BW15 (Northern Europeans), BW22 (Japanese), DW3 and DW4. An unusual genetic type (BfF1) of properdin factor B occurs in 22.6% of pateints with insulin-dependent diabetes mellitus as opposed to 1.9% of the general population.[14] The genetic focus for properdin factor B (BF) is closely linked to the HLA genes. This suggests that there may be a gene that predisposes individuals to development of insulin-dependent diabetes mellitus. Despite the even stronger influence of heredity on the susceptibility of individuals to adult-onset diabetes compared to insulin-dependent diabetes, no correspondence with HLA genotypes has been found.

Adult-onset diabetes mellitus

The loss of B-cells and severe impairment of insulin secretion that occur in insulin-dependent diabetes mellitus point to the pancreas as the site of major abnormality in this form of diabetes.

In contrast, adult-onset diabetes mellitus appears to represent a heterogeneous group of disorders in which both abnormalities in pancreatic endocrine secretion and impaired target tissue responses to insulin are involved. Although circulating concentrations of insulin can be low, normal, or high in adult-onset diabetes, abnormalities in the secretion of insulin from B-cells are usually demonstrable. One of the hallmark signs of adult-onset diabetes is the inability of sudden increases in plasma glucose to elicit a rapid burst ("first phase") of insulin release.[15] Although rapid infusion of glucose is a poor stimulator of insulin release, the acute release of insulin elicited by other known stimulating agents, such as secretin[16] or isoproterenol[17] may be entirely normal.

Target tissue defects in diabetes

The most common mechanism for target tissue insensitivity to the actions of insulin in adult-onset diabetes mellitus appears to be a decrease in insulin binding to target cells.[18] The decrease in insulin binding is due to a decrease in the number of insulin receptors per cell.[19] A few patients have been described with diabetes mellitus and acanthosis nigricans who have circulating antibodies directed against the insulin receptor.[20] Another group of female patients with diabetes, acanthosis nigricans, and ovarian dysfunction have been found to have a decreased number of insulin receptors on circulating monocytes. Bar et al. also reported a single case of a woman with diabetes and acanthosis nigricans, apparently normal insulin receptors, but insensitivity to insulin as a result of "postreceptor defect."[21]

Recently Tager et al. have described a patient with adult-onset diabetes and normal sensitivity to exogenous insulin despite high circulating insulin levels.[22] Study of insulin purified from extracts of this patient's pancreas revealed an abnormal amino acid sequence, with substitution of leucine for phenylalanine at position B-23 or B-24. This insulin did not bind as well as insulin receptors and did not elicit an increase in glucose uptake by target cells as effectively as normal insulin.

Insulin receptor antibodies, abnormal regulation of insulin receptor concentration on cell membranes, and genetically abnormal forms of insulin appear to be uncommon phenomena in patients with diabetes mellitus. Nevertheless, the clarification of these pathophysiologic mechanisms in a small number of patients emphasizes that different biochemical abnormalities can all produce the clinical syndrome of adult-onset diabetes mellitus.

What is the primary cause of tissue resistance to insulin in the majority of adult onset diabetes, who secrete normal insulin and do not have circulating antibodies directed against the insulin receptor? Two major hypotheses have been advanced. One hypothesis holds that the primary defect is in the target tissues. Since obesity is known to decrease insulin receptors, and many adult-onset diabetics are obese, the decrease in insulin receptors seen in these diabetic patients could be due to obesity. The differences between "normal" obese people and obese diabetics would presumably depend on the limited ability of the pancreas to increase insulin secretion in the diabetic group. The studies of Robinson et al[19] indicate that even non-obese diabetic subjects have a decreased number of insulin receptors on erythrocyte membranes.

The second hypothesis used to explain tissue insensitivity to insulin in adult-onset diabetes maintains that the insulin receptor changes noted in both obesity and diabetes are the result of the increased levels of circulating insulin that usually occur during at least some stage of the disease. Although the acute release of insulin in response to meals may be diminished or absent in mild, adult-onset diabetes, one to five hours after meals there is often a large overshoot in the amount of insulin released.[15] The "down regulation" of insulin receptors in target tissues that occurs in response to higher circulating insulin levels may lead to decreased insulin binding and apparent insensitivity to insulin's effects.

Pathogenesis of Diabetic Complications

Despite the different pathophysiologic abnormalities that can produce diabetes mellitus, all patients with diabetes mellitus are at risk for development of the chronic complications of diabetes—retinopathy, nephropathy, neuropathy, and accelerated atherosclerosis. The actual disease processes that lead to these lesions are not yet understood. However, since the genetic predisposition to juvenile-onset diabetes and adult-onset diabetes appears to be different, it is unlikely that any single, still-undetected, genetic abnormality can account for the chronic complications of diabetes. This suggests that one or more of the metabolic aberrations that result from inadequate insulin effects in target tissues are responsible for the long term complications of diabetes.

Hopefully, better means of restoring the normal actions of insulin in diabetic patients will be developed in the near future. This will probably require different strategies for the different forms of diabetes. Greater understanding of the diverse pathogenetic processes that can cause diabetes mellitus is essential for future improvements in therapy.

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AM *Controversies*

Keeping Costly Medical Care Affordable

Laurance B. Nilsen, M.D.

What is the role of HMO's, IPA's, and Foundations for Medical Care?

Tired of hearing this? Maybe so, but despite today's problems with inflation and energy, there still is very real public concern about the increasing cost of medical care and the dearth of effective solutions. Whether medical costs are considered in terms of the bills received by the individual patient who pays out of pocket, or in terms of the total cost to employers and industry who eventually pay most medical bills, the conclusions are the same: medical costs are fast approaching the limits of available financial resources. No matter that these increased costs are largely a result of better technology, greater demand, general inflation, and the predations of malpractice lawyers. Industry and other third parties who pay most of these costs have criticized medicine's role: a) we are often eager to adopt new technology on a large scale before efficacy or costs are fully considered. b) medical costs are not always proportional to quality when measured in terms of effectiveness, outcome, or morbidity. c) when insurance minimizes the patient's out-of-pocket costs, there is no incentive for either patient or doctor to limit utilization. The patients who are (or think they are) sick want the full benefit of moderm technology, and their physicians are understandably more concerned with prompt diagnosis and thorough treatment than with the insurance intermediary's financial status.

Given these observations, can necessarily costly medical care remain affordable in the absence of still more federal financing and regulation? The solution requires some means of controlling the demand for services and utilization that does not impair the quality of health care.[1] The Health Maintenance Organization (HMO) is one method of controlling utilization. Cost over-runs due to unexpected increases in utilization of medical services are absorbed by the HMO itself acting as the insurance intermediary; member doctors whose livelihood depends on a fiscally sound HMO are thus provided strong incentives to pare utilization down to essentials. Eliminated will be all of the procedures and hospitalizations that are not absolutely necessary but that are often undertaken for patient reassurance and satisfaction. Such "risk sharing" of HMO operating deficits by the member doctors is an essential feature of all HMO's whether they be closed panels (like ABC-HMO) or open membership IPA's (Independent Practice Associations).

IPA's are HMO's with membership open to any physicians in the community whose practice habits are sufficiently cost-effective and efficient to meet IPA standards. If the costs of medical services provided by the IPA exceed its prepaid premiums, the member physicians agree to absorb a proportionate share of the deficit by accepting substantially less than their usual fees from IPA patients as full payment. Once the IPA can cover the deficit and balance costs with premium income, the IPA doctors will receive full reimbursement up to the agreed upon limits. Although the IPA will have reinsurance against losses greater than 120% of premium income, the first 15% to 20% of the deficit will

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From: 3411 North 5th Ave., Phoenix, AZ 85013.

be absorbed by the IPA physicians. Cost over-runs have been common to all IPA's in their formative years, therefore a successful IPA will require strict utilization review and precertification for elective procedures and hospitalizations. The IPA cannot afford to carry member physicians who are indiscriminate in their decision to hospitalize or in their selection of procedures and consultations. IPA peer review is severe, for if a physician repeatedly refuses to alter his practice habits to conform with the standards of the peer reviewers and the community, he would either be assessed a surcharge or dismissed, leaving his IPA patients behind. Except for special circumstances the IPA patient (like any HMO patient) receives no insurance coverage when obtaining care from non-member physicians.

What else is needed for an IPA to succeed financially? All its subscribers will be required to see a primary-care IPA physician initially for all illnesses. These generalists in turn will be responsible for determining whether or not an insured patient will receive additional specialty consultation, and will have much of the responsibility for assuring that medical costs do not exceed IPA premium income. A successful IPA furthermore will require competent underwriting in order to set realistic yet competitive premiums: premiums significantly higher than other health insurance plans will simply attract subscribers who intend to utilize services, but unrealistically low premiums will lead to continued operating deficits that would risk widespread resignation of the member physicians. High utilization and costs by any one specialty group in the IPA will result in higher subsequent premiums for the entire IPA regardless of the number of "risk pools" the IPA subdivides between its generalists, specialists, and geographical areas. Finally, a successful IPA will need hospitals participating somehow in "risk sharing". Without such cooperation rising hospital charges may cause deficits in the IPA which must be offset by premium increases beyond any increases needed to cover the utilization of physician services. A relentless increase in premiums could reach the point where the IPA could not compete with closed panel HMO plans that had their own cooperative hospital arrangements.

How else can medical insurance be affordable? For ten years the Maricopa Foundation for Medical Care has provided insurance plans to employees and employers that cover essential medical needs and preserve the patient's freedom to select any physician he or she wishes. Foundation plans have been marketed at competitive premiums that are often lower than HMO premiums for comparable employee groups. Utilization review in the Foundation is as effective as that of any successful IPA in the country in terms of average length of hospital stay and total number of hospital days utilized. Most important, Foundations offer the patient a genuine alternative to the HMO concept. Whereas the HMO (or IPA) requires the patient's own physician to assume the role of "rationer" of medical services that are all virtually prepaid, the Foundations rely on both careful peer review by physicians and greater patient participation paying for discretionary services that are not absolutely necessary.

Could the Maricopa Foundation be more effective if it developed its own county-wide IPA? Business and industry want an IPA, believing as they do that IPA's and HMO's will ultimately be more cost-effective than Foundation plans. Because HMO's and IPA's must absorb the cost over-runs they are very attractive options to the large employers that self-insure. Patient's may desire an HMO because of their very low out-of-pocket co-payments, the convenience of "full service" under a single roof, and the implied promise that good health will be "maintained" as a result of the HMO annual examinations. Finally, many physicians want an IPA because of the need to compete with the ever-enlarging HMO's. In Maricopa County the two federally qualified closed-panel HMO's now cover 100,000 lives, equalling the present Foundation coverage. Federal legislation further requires all businesses with 25 or more employees to offer a federally qualified HMO by 1980. There is also concern that in the near future the government will give Medicare patients financial incentives to obtain medical care through IPA's or HMO's.[2] Those patients who are presently burdened by the out-of-pocket costs to pay the 20% co-insurance and the difference between their physician's charges and Medicare reimbursements may well opt for health care through an IPA or HMO. As medical costs continue to rise, there would be a point for each patient where the question of what was affordable would outweigh the question of which plan provided better care.

Despite persuasive reasons for starting a county-wide IPA, the following points should be considered: HMO's throughout the country usually enroll the initial 10%-12% of the eligible population. To grow further the HMO's will have to keep costs in line and patients satisfied. The costs of HMO's or any other prepaid plan will be lowest when most of the subcribers are healthy and do not require repeated hospitalization. The publicized savings of the HMO's will become harder to achieve as they enroll a greater proportion of subscribers who have complex illnesses that require extensive care and hospitalization. It is uncertain whether an IPA or HMO can provide care to such populations without eventually requiring federal subsidization and the control that inevitably follows.

Wrongly or not, industry anticipates larger savings with IPA or HMO medical insurance, and these employers will promote the plans to their employees. The extent to which employees accept the HMO option will be influenced by their previous experiences with traditional fee-for-service medical care but will ultimately be determined by any large cost differences existing between the insurance plans offered. To date, Foundation premium costs to the employees are not significantly different from HMO insurance. The question is whether this can continue.

The Foundation is progressing in the antitrust suit initiated last year by Arizona's Attorney General. Federal court has ruled that the Attorney General must first prove their allegations that the Foundation activities have inflated medical costs or otherwise harmed the community, and a trial will lead finally to the recognition that the Foundation has actually helped constrain rising medical costs in Maricopa County. The Attorney General has appealed this ruling, and it may be 18-24 months before even the question of a trial can be resolved. If the Foundation can continue to operate as it has been, it will remain a competitive option to HMO insurance.

Where does all this leave the Foundation in regard to the question of forming a county-wide IPA? Consider that a successful IPA would preclude open physician-membership and would require selection based on each physician's efficiency and cost-effectiveness. Consider that utilization review would be more stringent under the IPA. Consider that the IPA plan would restrict the patient's freedom to choose physicians. The Maricopa Foundation is unique among foundations in this country, for its size and effectiveness provide the competitive advantages of an IPA without requiring member physicians to share additional risk. Therefore the Foundation does not plan to start a county-wide IPA at this time. However, when it can no longer remain competitive with closed panel HMO's and other health insurance plans, the Foundation will have no alternative than to develop an effective IPA.

What about the larger question? Can Foundation, IPA, and HMO efforts to keep medical care affordable also keep the government from becoming even more involved than they presently are? Our best hope may be the very competition that naturally exists between these different delivery models. True competition will provide the incentives needed to constrain costs on a meaningful scale while leaving the responsibility for preserving quality medical care in the hands of physicians.

One final thought for those physicians intending to join an IPA. Traditional fee-for-service medicine can compete with HMO's for the care of large groups but only if the insurance premium for our services remains competitive. To achieve this the participating physicians will have to accept some form of reimbursement limits and strict peer review. These vexing constraints are well known to all of us within the Foundation, and will be essential requirements in any newly formed IPA that hopes to succeed. There should be no illusions that these constraints will become any less burdensome than they already are simply by joining an IPA.

References available upon request.

On HMOs

Donald F. Schaller, M.D.

Will Rogers once said "It's not what you pay a man but what he costs you that counts." Rogers' wisdom when applied to the arena of health care and its impact on our nation's health insurance premiums, personal finances, employer costs, and the gross national product would be dramatic. Rogers' "man" clearly rounds out as the one individual who impacts the greatest on the utilization of all health care services—the physician. Is the physician not the captain of the ship? Does he not define the work-up, schedule the surgery, order the hospitalization, write the prescription, request the consultation, write the order, supervise the rehabilitation, and on occasion, certify the cause of death. The equation encompassing the total cost of health care to the nation is a function of two important multipliers that must be considered as integral to the formula. The unit price or cost times the frequency with which the service or product is ordered and delivered provides the two variables that must be considered in any cost effective or cost containment measures.

Can any physician practice cost effective medicine without integrating his knowledge of both multipliers, the unit cost and the frequencey of delivery?

Too often we as physicians may neglect or fail to consider one or the other of these important multipliers in our patient's cost equation. How simple it is to justify or ignore these factors in the interest of "quality health care."

Can there be any doubt in the minds of any of us that our days of blissfully ignoring these important elements of the equation are ending? Read the Kennedy Bill, the Ullman Bill, the Long-Ribicoff Bill, the Durenberger Bill—their messages are clear. Our future habits are very likely going to be subject to much change. The legislative mandate will very likely pave our path with regulations and forms. Our actions through CHAP, Peer Review, Utilization Review, and other mechanisms may moderate the process but only time and our results can answer our concerns.

The HMO represents a response to the concerns of Congress and others. Its roots lie in China, the Grand Cooley Dam, Kaiser, Ross-Loos, and other places and times. Its trunk began with Public Law 93-222 (The HMO Act of 1973). Its name was conceived in the ideations of Elwood (Dr. Paul Elwood, InterStudy of Minneapolis). Does anyone believe that the HMO can

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From: Arizona Health Plan, Inc., P.O. Box 5000, Phoenix, AZ 85006. (Dr. Schaller is the Medical Director.)

truly maintain the health of any citizen? The title I think describes a concept with inherent incentives; not an objective or an outcome of the system.

The Law (Public Law 93-222) provides the definition which incites the objectives, the structure, the incentives and the risks.

Specifically, an HMO is:

1) An organized system of health care delivery. The term organization implies structure, management and all the elements requisite to providing care. . . hospitals, physician offices, nurses, paraprofessionals, educators and facilities, that provide . . . .

2) a comprehensive set of health care benefits. The benefit structure is generally more comprehensive than idemnity plans for a lower premium and results in a lower total out-of-pocket expense for the patient.

3) These benefits are provided to a voluntarily enrolled group of individuals. The adjective voluntarily is important here; it means that all members are enrolled in the HMO because they choose to be (over alternative coverage). Dual choice is a part of the Federal mandate and certainly is the preference of most HMOs.

4) Payment is through a fixed, prepayment amount plus an out-of-pocket co-payment amount. The deductibles are minimized. This is perhaps the most important implication for physicians (in HMOs) and their patients. Prepayment of health care costs absolutely dictates that the HMO organizational structure, physicians, and administration participate in the planning and budgeting process to predetermine all health and sick care costs, including out-patient visits, minor and major surgery, hospitalization, emergency care, lab and diagnostic studies, consultant costs, ambulance, dietary, optometric, mental health and pharmaceutical costs. These costs must be determined for the present and for the future annual period of the insurance contract (prospective budgeting). The purpose of this is to set a premium which assures both viability of the HMO and provision of services to the members.

The key to the organization structure and success of the HMO is risk — financial and other — that is borne by the HMO and its physicians. Clearly, the fixed prepaid amount by the patient shifts the financial risk from the patient to the system (HMO).

Suddenly, the incentives have changed. The incentive is now on the physician and the system to find out what Roger's "man" costs you. There is an incentive on the part of the physician to make all of the medical decisions:

1) What treatment, surgery, or referral is appropriate?

2) Which hospitalization should not occur and what would it cost and what is the risk of not providing the hospitalization?

3) Which medication is best not ordered?

4) Which consultations are the best for the patient and not just a response to patient pressure or malpractice concerns.

The impact of HMOs in Arizona and across the country grows. Recently a report to HEW showed that there are 217 operational HMOs. Ninety-six of these were developed with federal funds but 121 were developed solely with private funding. The two in Maricopa County are among those that are not federally-funded.

The developing HMOs then with their structure, shifting risk incentives, the economic and the budget process provide several significant philosophical shifts which impact on the health care equation. These include:

10 The use of the primary physician concept where most, if not all, medical care decisions about treatment, referrals, hospitalization and other items are made clearly by the physicians.

2. Hospital utilization and therefore overall hospital expenses are generally less in the HMO. The administrative costs of the HMO then can be looked at as one of the factors impacting on the decreased hospital utilization.

3) The HMO structure and its impact on the somatizing patient clearly brings this patient to an administrative level of concern at a more rapid rate than can possibly occur in the fee-for-service system.

4) The usage of health care coverage leverage to induce better patient compliance can have a significant impact on the overall outcome of alcoholism, drug abuse, and the acting-out patient.

5) The common chart can be a significant factor in helping to prevent repetitive diagnostic studies that may harm the patient and provide no real benefit to overall care.

6) The potential for integrated programs is greatly enhanced in the HMO. Examples of these are the 24-hour Urgent Care Unit coverage, patient education programs, the provision of ancillary services. Further, the better integration and utilization of the health evaluation process makes adequate follow-up a more likely event.

7) The defined prepaid population provides a significant and usually outlined data base for research and statistical performance evaluations.

8) The combined provider and insurer risk tend to produce incentives that insist that overall costs be acknowledged in the provision of services.

In summary, the HMO with its structure, the shifting risks, the incentives, and its budget process, can provide several philosophical shifts which can impact strongly on the total health care equation — unit price times utilization frequency. The impact is clear; the impact will grow. To dismiss this approach as simply another bureaucratic of federal intrusion is wrong. Better yet, consider in detail the risks, the incentives and learn how to apply those risks and those incentives to your own practice for better health care for your patients.

Independent Physician Associations

Bruce H. Shelton, M.D.

This paper is a discussion of Independent Physician Associations (IPA's); why they are necessary and how one IPA, Valley Care, intends to implement its programs.

The cost of medical care is rising at such a rate that it is becoming intolerable to the economy of the United States. The medical profession is being faced with a very hard choice in light of this cost rise; to either try and effect a change in the system ourselves or let our patients, through the hands of the government, force a change on us.

Let us briefly review the facts of this situation. Ten years ago 4.7% of the gross national product of the United States was spent on medical care. This year the propotion for medical care will approach 10% and the rate of rise is exponential.

Doctors can bang their fists on the table and stamp their feet on the floor against government control because they know what's best for their patients, but in another ten years when we ask our patients to spend between 15% and 20% (and most likely before ten years), patients are going to cry back to us; "We can't afford what you are doing for us and just maybe doctors don't really know what's best."

Reference is made to "our patients," as it isn't really the big bad government in Washington that is going to control doctors; it's patients who will instruct their government to do something about the problem. The question, therefore, facing physicians is no longer change or not change; it's what kind of change and who will direct it?

The medical profession should effect a change in this admittedly costly situation, as physicians really do know what's best for their patients. Physicians should run the show rather than a faceless bureaucracy in Washington that has the proven ability to turn medical care into an Army outpatient clinic that most physicians have had the pleasure of working in at one time or another.

What then is the basis for the problem that needs to be solved?

The problem is that none of us in the health care equation (doctors, hospitals and patients) have any *incentive* under the present system to reduce costs!

It is estimated that 85% of all the people in this country have fairly comprehensive health care benefits. For these people medicine is essentially a free lunch. They come to our offices and hospitals and turn themselves over to us without question of cost.

The other 15% are our old and/or poor that get taken care of also, after crossing some rather high barriers to care, but they do get cared for.

What incentive does a patient have to leave the hospital a day earlier rather than a day later? Insurance covers the cost.

What incentive does a doctor have to discharge a patient a day earlier rather than a day later? He gets paid more for the extra day and insurance covers the cost.

What incentive does a hospital have to encourage early discharges? If you harass your medical staff, they will take patients to other hospitals and shorter stays mean less income and the inability to build another wing.

One simple solution to the problem would be to reduce the comprehensive insurance benefits that patients have. If medicine didn't seem *free* to them and they had to actually pay the bill (they do now in an indirect manner, but they don't realize it), patients would become cost conscious medical consumers and force economy on the system.

Faced with the choice of an inpatient or outpatient hernia repair a lot of patients would choose outpatient if they had to pay the difference. They would also choose to go home that one or two days earlier from the hospital if they had to pay for the extra costs.

Patients would also question very closely what we doctors do and make us practice more economically. Given a change, they would *seek out and find* the doctors who charge $15 for an SMAC rather than the doctor who charges $100 (some doctors still do it at $5 per test in their office rather than send it out).

Doctors and hospitals would probably become very cost effective very quickly if patients became cost conscious, but patients, politicians and government will never let their hard-earned comprehensive benefits be taken away from them. They are demanding more rather than less benefits because they need protection from the medical profession because it has *no incentive* at present to reduce costs.

One other solution would be for doctors to set themselves up in such a model that quality cost effective medical care would be rewarded when less is done rather than more.

Let's face it, there are doctors in this community that hospitalize less than other doctors and get just as good a result, if not better, than doctors that use more hospital beds. There are doctors who use a $15 SMAC rather

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From: Valley Care, 2525 West Greenway Road, Ste 300, Phoenix, AZ 85023. (Dr. Shelton, Medical Director).

than a $100 SMAC.

There are doctors that do a good comprehensive quality physical exam, including lab and x-ray, for $125 rather than $325.

There are surgeons who can discharge a gallbladder surgery patient in 4 days with an excellent result, rather than 8 days.

There are gynecologists who have a lower hysterectomy rate than others without increased morbidity.

There are primary doctors who keep late office hours so that their patients don't have to go to the costly emergency rooms with simple calls.

There are ENT men who don't take out every other tonsil that come into their office.

There are doctors who insist on using outpatient surgery facilities regularly.

If only a complete spectrum of the *naturally cost effective* doctors could be put together as a team. A premium could be calculated for their *normal fees* and as a collective group, they would be lower in price than the rest of the community and would attract more patients *without a change* in their quality of care.

This is the philosophy of the Valley Care IPA. To assure the plan's success the following checks and balances will be instituted:

1) Every physician in the plan has an equal vote.

2) Physicians can be dropped from or added to the plan by majority vote of the member physicians (with proper protection for appeal).

3) Each physician agrees that primary care physicians are the proper entry into the system and specialists will not see patients unless referred by a primary physician.

4) Two separate risk pools will be established, one for primary doctors and one for specialists.

5) The primary physician will be charged for *unnecessary* overutilization of specialists and hospital.

6) Specialists will be charged for *unnecessary* overutilization of hospitals and procedures.

7) Primary physicians and specialists will also be individually charged for *unnecessary* underutilization if a delay in a needed procedure or referral eventually costs money (i.e. if a primary doctor splints a fracture that should have been casted and the delay eventually causes a surgery to repair a nonunion, had it been timely referred, the cost belongs to the primary risk pool).

8) Necessary overutilization, such as a valid flu epidemic or an outbreak of valid hot appendicitis, will be shared equally.

9) Utilization committees compsed of primary and specialist physicians will determine what is necessary or unnecessary with an appeal system to an outside agency, such as the University Medical School.

We feel that by starting out with cost conscious quality physicians and then fine tuning their medical care with the best type of peer review (a group of 30-35 physicians will all know each other), a system will be established where doctors will be rewarded by doing less but will also be penalized for not doing enough.

We see our IPA as a citywide association of groups of physicians of 30-35 in each hospital location as sort of sub-group IPA's within one big IPA that can sell its services uniformly to employers who have employees living all over the city.

Each sub-group IPA will have its own separate primary and specialist risk pool, so in a sense each sub-group will also be competing with the other area sub-groups.

The entire IPA, which will be called VIP (Valley Independent Physicians), will contract with Valley Care, an HCSO (Health Care Service Organization), which is a non-profit corporation operating under Arizona law and is a quasi-insurance company in that it has dollar reserves and sells the plan's programs.

Each sub-group within the IPA will be headed by a local medical director elected by his peers and will serve on the HCSO Board of Directors. The medical director will be reimbursed for his time and services.

No one within either the HCSO or IPA will own a pecuniary interest in the plan that can ever be sold for a profit. The only way that anyone can make money is through the successful practice of medicine or payment for valid rendered services tied directly to an acceptable hourly rate.

This system will enable us to compete with the high prices in the private sector and also with the group practice HMO's that presently captivate over 10% of the patients with insurance in Phoenix.

We feel that we will have better *patient acceptance* than the group HMO's (probably the best lay translation for quality medical care there is).

We feel that one big IPA (as proposed by the medical society) will not be as effective as our system, as it will include every doctor in town (overutilizer and underutilizer) which will negate most of the positive advantages of our plan (we don't want our office calls slashed by 30% because the plan has a gynecologist who takes out every third uterus).

We will be able to keep our prices where they are now but will be able to expand our practices so that more partners can be brought in to reduce our share of the overhead so that our next price increase can just match the inflation rate rather than 1-1/2 times it, as it is now.

If we can keep the cost of medical care at just under 10% of the GNP and have our costs grow at the same rate as the economy, the government will leave us alone. It is the disproportionate rate of growth in our costs that is going to cause socialized medicine, which will be the worst thing that ever happened to medical care in this country.

The IPA is the last chance that we, the doctors, have to change the system ourselves.

Banging our fists, stamping our feet and burying our heads in the sand won't make the problem go away. The choice really is ours!

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H.M.O.'s and I.P.A.'s:

Vehicles of the Future?

Increasing expense of medical care in the United States has resulted in the development of methods for cost containment. One such approach is prepaid medical care in the form of a Health Maintenance Organization (HMO). These organizations seem to be initially cost-effective because they place certain limitations on the availability of medical care (see article by Dr. Schaller page 773). As an alternative to these structured groups of physicians the concept of a "HMO without walls" has evolved and is now being actively discussed within the Tucson and Phoenix medical communities. Basically, in an Independent Physician Association (IPA) physicians will share in certain risks and will agree to accept as payment in full the fees provided by patients who join this association (see article by Dr. Shelton page 775). Although this concept is somewhat novel, it still, in my mind, does not deal in a longterm way with what appears to be the goals of the federal government, that is, a prepaid health care system. Although the titles HMO or IPA may gender some sort of concept of independence from federal control and regulation I believe that we are really sticking our heads in the sand if we feel that such is the case. Obviously an HMO or IPA concept will serve as a vehicle, not as an alternative, for the introduction of significant cost containment processes. We as physicians will have little to do with accepting or negating these proposals as they are presented for we will be basically technicians performing the work while others will be dictating what shall or shall not be done. The IPA is an interesting concept which should be closely scrutinized by all members of the Medical Association. It has some attractive features which could make our health care delivery more cost-effective. Obviously there will be those amongst our members who will feel threatened by such a concept but I think the alternatives are slowly disappearing. Most patients will ultimately be covered by some sort of third party carrier be it federal or private, and we will need to somehow gain access to those funds. The private insurance industry will be looking for more effective ways to diminish the cost of health care and will be looking to either HMO's or the other options such as IPA's and Foundations as a way to accomplish this task. Therefore, as a large precentage of the population falls under such regulation we will be forced to deal with them and this will no doubt lead to more active involvement by almost all physicians (see article by Dr. Nilsen page 771).

It would appear to me that we are in a testing phase whereby these various vehicles are being put out for inspection to see how they hold up under the stresses and strains of the marketplace. It may very well be that the HMO's will not succeed but that the IPA situation will. We live in a "free" society and as such must allow all alternatives a chance for expression. Keep in mind, that times are changing and what looks good today may be obsolete tomorrow. There may also be other alternatives which have not yet been put into use and we should carefully look at all of them as they come into focus to pick out the one which will be the most comfortable and enduring.

Marshall B. Block, M.D.
Editor

The Multiple Sclerosis Clinic of the University of Arizona Health Sciences Center

A demyelinating disease of unknown etiology, multiple sclerosis (MS) affects only humans and usually strikes during the prime of life. Many questions about MS need answers.

Why do some patients function well with minimal disability and at autopsy have tissues severely affected just as patients known to be disabled for years? Why do patients have exacerbations, and more importantly, what allows some to recover after an exacerbation while the disability of other progresses relentlessly without exacerbation? What influences in our environment cause MS and trigger exacerbatons? Do some have a hereditary predisposition to develop MS? Do some lack the defense against progression? Do some have the capacity to transmit nerve impulses through unmyelinated nerves? Can predisposing factors be identified and neutralized? If so, could the progression be arrested and ameliorated?

Epidemiologists *have* identified predisposing factors in MS. The geographic distribution may provide clues to the cause of the disease. Published rates show a direct relationship to latitude.

Prevalence rates are much higher than expected in the Orkney and Shetland Islands, and lower than expected in Japan. Migration studies in Isreal and the Republic of South Africa demonstrate an association of developing MS with the country of origin. Thus the hypothesis that a causative agent present in certain geographic areas leads to the disease after an incubation period of years following childhood exposure.

The reported 9.4% of MS patients with a family history is a frequency ten times higher than expected in the general population. This frequency suggests a hereditary factor in the causation. An association between MS and the genetically pre-determined HLA-A3, HLA-B7, and HLA-DW2 histocompatibility antigens further supports this theory.

Titers of measles antibody in the blood and CSF of MS patients are elevated. Leucocytes of MS patients fail to respond normally to measles virus and measles virus has been isolated from the jejunum of MS patients. However, while conclusive proof is lacking, these observations tend to implicate the virus or measles-like virus in the pathogenesis of MS. Since MS is not known to affect species other than humans, there is not a satisfactory experimental model. The MS patients thus must direct further productive research. Patients in a multiple sclerosis clinic can be continually prospectively questioned and frequently examined. Such a clinic also provides a resource for tissue samples and a reservoir of well-studied patients essential for education and ideal for therapeutic trials with promising agents.

Approximately 125 to 130 patients with probable or clinically definite MS are registered with the Multiple Sclerosis Clinic at the University of Arizona Health Sciences Center. Eighty age and sex-matched controls without neurological disease are also enrolled.

The monthly telephone or bedside interviews, performed by an experienced nurse practitioner, provides 30 day symptom scoring and prospective epidemiological data. Such close contact with patient and family also provides emotional reassurance. A three-month clinic visit, identifies subtle complications and records a Kurtzke neurological status evaluation. Serum specimens are collected and preserved for present and for future investigations. For a drug to be proven 50% effective in reducing relapses or the progression of disability the entire MS Clinic patient population has to be involved in a 4 to 8 year double blind crossover study.

The MS Clinic has been very valuable therapeutically, scientifically, and educationally. Using retrospectively accumulated data, exposure to animals has been shown to not be a significant factor in the causation of MS. Similarly, data from the Clinic reveals that trauma and local and general anesthesia neither cause multiple sclerosis nor precipitate exacerbations. The safety of influenza vaccination in the MS clinic population has been established. From retrospectively accummulated data, it appears that there is an association between familial MS and familial neoplasia of all types. The MS Clinic patient group has provided data disproving the value of MS associated antigen tests as an indicator of MS.

In summary, the MS Clinic provides a basis for more rational rehabilitation and symptomatic treatment of MS patients by determining the influence of various environmental factors on the cause of MS; provides a resource of patients and tissue samples for investigations; and provides a reserve of well studied patients for education and is available for therapeutic trials as promising agents become available.

Colin R. Bamford, M.D.
Assistant Professor of Neurology
Michael S. Smith, M.D.
Assistant Professor of Neurology
William A. Sibley, M.D.
Professor of Neurology
Louis J. Kettle, M.D.
Dean, College of Medicine

Auxiliary Highlights

Yuma County

County Medical Society Auxiliaries — that's where the real action is! And a lot of fun, too.

Let's take a look at the activities being planned by President Ann Tennant and the Yuma County Medical Society Auxiliary.

Cardio-pulmonary Resuscitation classes for the public will be taught for the third year by Auxiliary volunteers. Yuma owns adult and infant Resusci Annie dolls, has its own copy of the approved teaching film. More important, the Auxiliary has eight trained instructors—Lyn Webb, Lynn Darwin, Shirley Morris, Carol Christ, Marty Anderton, Leslie Cannell, Dee Sussman and Ann Tennant.

Classes in Breast Self examination will be offered again by the Auxiliary. A Reach to Recovery volunteer from the American Cancer Society will show a training film and give a demonstration on "Betsy" at Yuma's October meeting. Six Auxiliary members have volunteered to take this program to the community—Margarita Alvarez, Kusum Sud, Shirley Johns, Susi Injeian, Dee Sussman and Bernice Roberge. Yuma is optimistic about developing a BSE program in Spanish.

Health Careers Day for high school students will be sponsored again in the spring.

Members are completing the distribution of 10,000 immunization record booklets to physicians' offices and schools. The booklets are also available at the County Health Department's baby clinics and the hospital's newborn nursery.

The Auxiliary will assist the Hospital Foundation of the Yuma Regional Medical Center in its February fund-raising dinner dance. Proceeds will go toward the purchase of a CT Scanner. Barbara Winfield chairs an Auxiliary committee that will decorate the convention center for the party and also set up and decorate the tables for 800 guests.

Yuma's Auxiliary will have monthly business meetings, which will include programs of special interest to members. In January, for example, gymnstics teacher (and member) Sylvia Linden will give a demonstration on "Keeping Fit". Another member, Katherine Irwin, is an expert on desert flowers and vegetation and she'll give a slide talk on "Desert Beauty" at the March meeting. The February meeting will feature The Readers Theatre from Arizona Western College.

Yuma Auxiliary parties give doctors and spouses a chance to relax with friends and meet newcomers to the medical community. This year the traditional fall dinner party will have an Italian theme. At Christmas there'll be a dinner dance at the country club with the dentists and pharmacists joining in the fun. The annual family picnic, in May, will feature the usual good food and a variety of recreation—swimming, trampoline, horseshoes, volleyball, relay races.

Combining fun with purpose, this year the Yuma Auxiliary will also sponsor a fund-raising cocktail party. Money will go to the Nursing Scholarship which the Auxiliary awards each year. Talented members of the Society and Auxiliary will provide entertainment with guitar, organ, piano and disco dance selections.

Mrs. John T. Clymer,
President
ArMA Auxiliary

President
William E. Crisp, M.D.

President Elect
John T. Clymer, M.D.

Vice President
Clyde W. Kurtz, M.D.

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John W. Kennedy, M.D.
William B. McGrath, M.D.
John Stoner, III, M.D.
William C. Weese, M.D.
Donald J. Ziehm, M.D.

AM Correspondence

Continuing Medical Education

Editor—

Continuing medical education has become not only big business but also big busyness. Professors and practicing physicians are flying and driving all over the country in an effort to spread and absorb knowledge.

What kind of knowledge is being apparently spread and what kind of knowledge is being apparently absorbed? Are the professors genuinely interested in imparting and sharing newly acquired knowledge and are those in attendance genuinely interested in absorbing knowledge in this manner?

The cost of a continuing medical education course has become substantial and the dictum that the physician will impart his knowledge to each successive generation without financial renumeration has fallen by the way-side. The price to teach for having been taught seems to hold no longer true.

The rooms are semi-dark, the screen is semi-light, the lecturer is semi-awake and the audience is semi-asleep. A routine of verbage eminates from the rostrum mostly of detailed sophistication almost of no use to the practicing physician. The course was to deal with infertility—my pre-conceived (pardon the pun) notion was some information as to the diagnosis and treatment of this condition. The serial slides projected on the screen were a joy to all those interested in polychromatic transparencies. The colors ranged from beautiful riviera blues to fiery reds to avacado greens. Each slide, taken at 2 millimeter intervals, of the cervix of the infertile rat specially stained to reflect the glycogen content as it progressed from caudad to cephalad. I loved the colors but my infertile patient gained nothing and I gained very little. The oxygen content in the room decreased, the carbon dioxide content increased and the semi-awake state became progressively less semi and more total. Coffee and fresh air reversed the inertia.

The next speaker, a dynamic one, a boutinier in his lapel, a deep Lawrence Olivier voice - a ubiquitous problem, "The Alcoholic" was the topic. The introductory remarks stressed the inability to define "an alcoholic", the inability to state what is too much drinking and what is moderate drinking only to continually refer to the moderate drinker which the lecturer had just told us he could not define. Without definition of what he meant I sure did not know what he was talking about but I had come and paid for this.

The warning lecturer; the one with pounds of platitudes, the nonspecific specifist, the detailed generalist who speaks of high index of suspicion - translated that means "you had better think about everything." He goes on to speak of weighing disadvantages versus advantages. This is what everybody does all day long. Crossing the street provokes that decision. We hope to get to the other side - had we known we would get hit by a gasoline truck we would not have done so. How trite can one get. Keep side-effects in mind - who doesn't? On it goes for 45 minutes telling us not to make mistakes. As if we are about to embark on a planned mistake-making venture.

A continuing medical education course then is a lecture given in another city, at great expense in great discomfort for CME points about a subject that was recently well written up by the same speaker in a good journal, read at little expense in ones' own home in comfort and for no CME points but the acquisition of knowledge was more effective with the latter approach than the former.

R. Kirschner, M.D.

Cardio-pulmonary Resuscitation

Editor—

Cardio-pulmonary Resuscitation may have a place in the continuing education endeavors we now champion but I have a serious and deep seated objection to the stance now in vogue among the hospitals of Phoenix.

I have been notified, as every other physician has been notified, that I must take a scheduled "course" in CPR if I am to be granted privileges in those hospitals where I hold staff membership. This dictatorial directive is hereby challenged! I very much doubt that there is on record in any hospital or any where else the fact that this directive is free and clear of legal entanglements. Somewhere in the forest of those laws passed to protect us poor innocents I recall that capricious directives on an ad hoc basis which abrogate my constitutional rights to make a living in the profession I practice are **verboten.**

The hosptial administrators, eith innocently or by osmosis from on hig have convinced the medical staff th CPR is not only an absolute necessity b one that has been endorsed by sor presumably straight thinking expe now wandering around in the miasma health administration "for the good all." Some of our respected colleagu have aided and abetted this mandato requirement for staff membership. This directive is ill founded and qua fradulent which does not enhance t prestige of the participating colleagu despite the fact that the hospit administrators can probably produ meeting minutes to prove that there w "adequate staff approval" preceding t publication of the staff directive. If object to our friends being "front me for the hosptials then a "sit tight" attitu makes us all accomplices in this deplora situation.

The sermon I am preaching here is simple one:

If CPR is an inescapable way of life these times, let it be voluntary and n mandatory.

The CPR directive is to some degr fradulent as the teaching deliberate disregards the recent statements by D Heimlich that the controversy methodology should be stopp immediately. Furthermore, and greater importance, is the fact that t instructors of the CPR "course" fail give advice as to what should be done case the "**Heimlich Manoeuvre**" fail Tracheotomy becomes an imperati necessity. Who should perform tracheotomy? It seems logical to assum (if one really is an advocate of CPR as a li saving measure) that most everyo should be able to do a tracheotomy. If s it should be an addendum to the CP course. The hero of the "curbsi penknife" saga is known to all. How ma physicians carry a knife of any kind their person? Very few. How man physicians carry a large bore needle i their pocket to make a quick and eas airway in that part of the trachea inferi to the blocked voice box? Very few a cognizant of this life saving surgic airway and there are those anesthetis who say it is ineffectual because th internal diameter is always inadequat for a satisfactory ingress and egress of al How many physicians carry on the person the small tracheotomy instrume advertised in the Journal of the America Medical Associaton?

Despite my aversion and condemnatio of the CPR course I did take it. Believe yo me it is the last time I will expose myse to that kind of coercion. I found th "course" traumatic and tiring and th mannikins less than sanitary, far fro esthetic, and a very good way to pas along a virus or some other crud hiding i his "wet wipe and blow" method. The CPR is also more than an ordinary strain n a person under treatment for cardiac r respiratory disability.

Governmental bureaucratic encroachment on the practice of medicine is bewailed and bemoaned countless times each day in any physician bull session. Why should we be paradoxical in our ttitude by standing by in sheepish ubjucation just because some of our olleagues are the ones who are "doing us n"? By acceding to the hospital directive we are losing the hand hold on our destiny and we can ill afford to allow this o happen.

Now is the time for all good men. . . . .

Dermont W. Melick, M.D.

1959 - 1979
The Medical History Society of Arizona

Editor—

The Medical History Society of Arizona celebrates its 20th Anniversary year. In honor of the occasion, a special lecture entitled "History of the Barrow Neurological Institute" will be presented by Dr. John Green, one of Society founders, on Tuesday, October 30, 1979, at 7:30 p.m. at the Arizona Medical Association building, 810 West Bethany Home Road, Phoenix.

On January 16, 1959, a meeting was held at the home of Dr. and Mrs. Louis Jekel for the purpose of establishing an organization dedicated to the study of the history of medicine. In addition to the Jekels, other original members were Dr. and Mrs. Earl Baker, Dr. and Mrs. James Barger, Dr. and Mrs. John Green, Dr. and Mrs. John Kennedy, Dr. and Mrs. David Long, Dr. and Mrs. Paul Matte, and Dr. and Mrs. Howell Randolph. Both physicians and spouses were active participants, taking turns in giving formal presentations several times a year. The group initially met in homes of members, as a formal dinner club, with Dr. Jekel as the first President and Dr. Barger as the first Secretary. As membership increased, the meeting site was changed to restaurants, to the offices of the Arizona Medical Association and, most recently, to the Maricopa County Medical Society. Over the years there was great diversity of topics discussed.

Of the founding members, Doctors Jekel and Randolph are deceased and Doctors Barger and Matte have moved out of state. Dr. and Mrs. Lewis Claypool, who joined the Society in 1960, were frequent gracious meeting hosts and worked hard to keep the fledgling society strong during its early years. In addition to Doctors Jekel and Claypool, the office of President has also been held by Doctors Howell Randolph, John Alsever, Imre Sandor, John Green, Edward Sattenspiel, James Fillmore, Maurice Richter, Jack Wilson, Earl Baker, Stanley Millstein and Clifford Ernst, with the current President, myself, elected in 1977.

As the Medical History Society of Arizona enters its twenty-first year, the October, 1979, meeting will be set aside in tribute to, and memory of, the persons who have supported it so strongly. I will briefly review the highlights of the first twenty years, and Dr. Green will narrate the history of the Barrow Neurologic Institute, of which he is also a founding member. The public is cordially invited.

William C. Weese, M.D.

ArMA Reports

THE MINUTES APPEARING IN THIS SECTION HAVE BEEN EDITED TO CONSERVE SPACE. A COMPLETE COPY OF THE MINUTES OF ANY MEETING WILL BE MAILED TO ANY MEMBER REQUESTING THEM.

EXECUTIVE COMMITTEE

The meeting of the Executive Committee of the Arizona Medical Association, Inc. held at 810 W. Bethany Home Road, Phoenix, Arizona, on Friday, August 3, 1979, a quorum being present, convened at 6:34 p.m., William E. Crisp, M.D., President and Chairman, presiding.

Department of Health Services - Activity Report

Dr. Dandoy reported to the committee on the recently-discovered problem with infant formula, as well as the ongoing pesticide situation, advising the membership that the Environmental Protection Agency had determined that it would be doing the pesticide control in the state and had already scheduled a 3-day hearing for the purpose of obtaining all testimony prior to making decisions as to the handling of the matter.

Dr. Dandoy advised that Lewis Kurke, M.D., a psychiatrist from the State of New York, had been hired as the new Assistant Director for Behavioral Health Services to coordinate all mental health and drug abuse problems. Additionally the matter of the termination of the Health Planning and Development Agency on July 31st was discussed, with Dr. Dandoy stating that the termination had come about by reason of a lack of funding through Congress, that the personnel from that agency had been interwoven into other departments, with the hope that funding would eventually become available for the reopening of that agency. Likewise, the anticipated curtailing of about one-third in all federal funding, would undoubtedly require limitations of other departments in the near future.

Podiatrists and Hospital Privileges

Mr. Jacobson reported to the committee on recent meetings held by counsel for the podiatrists, the Arizona Hospital Association and himself, on behalf of the Association, relative to the podiatrists current attempt to obtain admission to hospital staffs throughout the state. Following a lengthy discussion of the matter,

IT WAS MOVED AND CARRIED THAT THE ARIZONA MEDICAL ASSOCIATION, THROUGH AN ARTICLE IN MEDICAL MEMOS REAFFIRM ITS PREVIOUS POSITION TAKEN BY WAY OF A JOINT RESOLUTION WITH THE ARIZONA HOSPITAL ASSOCIATION, WHEREIN IT ENCOURAGED HOSPITALS TO CONSIDER QUALIFIED PODIATRISTS FOR STAFF ADMISSION, AND TO ADVISE THE MEMBERSHIP THAT, SHOULD THEY DESIRE FURTHER INFORMATION OR ASSISTANCE IN RELATION TO THIS MATTER, IT WOULD BE AVAILABLE TO THEM THROUGH THE ASSOCIATION OFFICES.

Survival Guide for Telephone Directory

The committee discussed the request received from the Arizona Medical Association Auxiliary, Inc. relative to providing a letter to Mountain Bell supporting "Survival Guide for Telephone Directory."

IT WAS MOVED AND CARRIED THAT THE ARIZONA MEDICAL ASSOCIATION, BY LETTER, ADVISE MOUNTAIN BELL THAT IT SUPPORTS THE INCLUSION OF A "SURVIVAL GUIDE" IN THE TELEPHONE DIRECTORIES IN THE STATE OF ARIZONA.

National Center for Health Services Research

The committee received and considered the July 31, 1979 letter from the Arizona State Senate requesting support for a grant proposed to the National Center for Health Services Research. It was the consensus of opinion that sufficient documentation was not presently available and that a request should be made to the Arizona State Senate and/or the University of Arizona Health Sciences Center for a copy of the grant for review and use by the committee prior to its determination in this matter.

Review of Board of Directors' Agenda for 8/4/79

The Board of Directors' agenda for August 4, 1979 was discussed, with additions and changes being made.

Other Business

Membership Activites

Gary L. Barnett reported to the committee regarding the recent offer of the AMA to assist the Association in membership activities, advising that, in fact, an AMA representative had already been to Arizona, met with the Maricopa and Pima County Medical Societies, and was now in process of formulating recommendations and programs for consideration by the Ad Hoc Membership Committee.

Industrial Commission of Arizona

Mr. Robinson presented to the committee a recent article contained in *Medical Leadership News* relative to the recently issued fee schedules by the Industrial Commission of Arizona, with Mr. Barnett giving a brief statement as to conversations he had had with the ICA subsequent to the release of the article.

Dealing and Working Effectively with the Media -Workshop

Dr. Clymer, on behalf of the Arizona Medical Association Auxiliary, Inc., presented to the committee a request that ArMA and the Auxiliary co-sponsor a workshop relating to dealing and working effectively with the media. It was anticipated that this would be a day-long program to be presented by someone from the AMA staff sometime late this year or early next. Following discussion, IT WAS MOVED AND CARRIED THAT THE ARIZONA MEDICAL ASSOCIATION APPROVE, IN PRINCIPLE, THE HOLDING AND CO-SPONSORSHIP OF A WORKSHOP ON DEALING AND WORKING EFFECTIVELY WITH THE MEDIA AND THAT THE ARIZONA MEDICAL ASSOCIATION AUXILIARY, INC. BE REQUESTED TO FORMULATE DETAILS FOR PRESENTATION TO AND DISCUSSION WITH THE EXECUTIVE COMMITTEE AT ITS NEXT MEETING.

BOARD OF DIRECTORS

The meeting of the Board of Directors of the Arizona Medical Association, Inc. held at 810 West Bethany Home Road, Phoenix, Arizona, on Saturday, August 4, 1979, a quorum being present, convened at 10:04 a.m., William E. Crisp, M.D, President and Chairman, presiding.

Board of Directors

BOMEX Appointments

Reappointment by the Board of Medical Examiners of James E. Brady, Jr., M.D. for a second term ending 2/26/84. RECEIVED.

The Board also noted that the vacancy which had been created by the termination of the term (6/30/79) of Manus R. Spanier, M.D. had not yet been filled.

Litigation Update

Edward Jacobson, general counsel for the Association, reported to the Board regarding the current status and appropriate law regarding each of the following. Federal Election Commission, Internal Revenue Service Audit, Federal Trade Commission Subpoena, Chiropractors, Podiatry, and the suit filed against the Attorney General. Following a general discussion, it was the consensus of the Board that the mailing contemplated by the action of the Executive Committee on August 3, 1979, i.e., a restatement of the joint resolution of ArMA and the Arizona Hospital Association regarding podiatrists, be enlarged to include all hospital Administrators and Chiefs of staff.

Status Report of House of Delegates Resolutions

The following status report of resolutions adopted in some form (not including resolutions not adopted, tabled, etc.) during the 1979 House of Delegates meeting. RECEIVED.

1) 1-79 - 1980 Calendar Year Budget for income and expenses - Finance Committee is in the process of determining individual committee budgets.

2) 2-79 - P.L. 93-641; Composition of HSA Board of Directors - Resolution No. 54 introduced in the AMA House of Delegates.

3) 3-79 - National Health Insurance - AMA advised of position - on other action necessary.

4) 4-79 - Medical Student Scholarships - no further action necessary until 1980.

5) 5--79 - The American College of OB-GYN Statement of December 1977 Regarding Abortion - no further action necessary.

6) 6-79 - Roman G. Mislicky Scholarship - first recipient - Sharon L. Sandell of Peoria.

Auxiliary Report

Annual Convention - Chicago

Eloise Clymer reported on the recently attended convention held in Chicago, which seven representatives fro Arizona were present. The meeting w very beneficial, with some of th highlights being the presentation of check for over a million dollars by th national organizaiton to the Nation Research Foundation; and presentatic to the Arizona Medical Associatic Auxiliary, Inc. of an award for the highe membership in the Western Regio States.

Future State Activities

Mrs. Clymer reported that some of th Auxiliary's priorities for the coming yea were legislative matters, health project public relations and communication recruitment and a leadership conferenc

Executive Committee

Membership Classification Change Approved

Maricopa County Medical Society:

1. M.W. Westervelt, M.D. - Active t Associate - Account Retirement - Due Exempt - Effective 1/1/79.

2. Thomas P. White, M.D. - Active t Associate - Account Residency - Due Exempt - Effective 1/1/79.

Mohave County Medical Society

1. Arthur A. Arnold, M.D. - Active t Associate - Account Retired - Due Exempt - Effective 1/1/79.

Pima County Medical Society

1. Sherwood Burr, M.D. - Active t Associate - Account Retirement - Due Exempt - Effective 1/1/79.

Yavapai County Medical Society

1. Carl R. Newman, M.D. - Active t Associate - Account Retirement - Due Exempt - Effective 1/1/79.

Benevolent & Loan Fund . Committee

Roman G. Mislicky Scholarship

Letter date 4/21/79 from Roger A. Lyon President, Valley National Bank regarding Roman G. Mislick scholarship. RECEIVED

1979 ArMA Medical Studen Scholarships

Medical student scholarships granted i 1979 to Toni Jo Brayer of Page, Stephe W. Coons of Tucson, Theresa Ann Culle of Phoenix, Lane P. Johnson of Prescott Rene O. Parker of Nogales, Jeffery A Poage of Sedona, Sharon L. Sandell o Peoria (Roman G. Mislicky Scholarship and Alan J. Webb of Scottsdale RECEIVED.

1979 Robert Carpenter Medical Studen Scholarship

Robert Carpenter Medical Studen Scholarship for 1979 awarded to Ric Weimar of Phoenix. RECEIVED

Finance Committee

Financial Statement for period endin 6/30/79. RECEIVED.

Governmental Sevices Committee

Proposed Workshop on Health Plannin

Following a review of the recommendation of the Governmental Services Committee made on June 20, 1979, and a brief report by Dr. Clymer concerning the discussion held at that meeting and the fact that the Auxiliary was willing to help coordinate the materials for and scheduling of a workshop on health planning for physicians who indicated a desire to attend,

IT WAS MOVED AND CARRIED THAT THE ARIZONA MEDICAL ASSOCIATION SPONSOR A WORKSHOP ON THE GENERAL SUBJECT OF HEALTH PLANNING, HOW IT WORKS IN ARIZONA AND HOW IT AFFECTS PHYSICIANS.

Legislative Committee

Letters of commendation from Maricopa Foudation for Medical Care and Mutual Insurance Company of Arizona re: the legislative work done by Associate Executive Director, Gary L. Barnett. RECEIVED.

Medical Education Commmittee

Accreditations Granted

St. Mary's Hosptial & Health Center of Tucson accredited for CME for two years, effective December, 1978. RECEIVED.

AMA Committee on Continuing Medical Education

IT WAS MOVED AND CARRIED TO NOMINATE ROBERT E. T. STARK, M.D., FOR APPOINTMENT TO THE AMA COMMITTEE ON CONITNUING MEDICAL EDUCATION.

Professional Committee

Homeopathic College of Medicine

Letter of July 10, 1979 regarding the establishment of the James Tyler Kent College of Homeopathic Medicine in Phoenix. RECEIVED.

Section on Cancer - Dissolution

The Board reviewed the recommendation of the Professional Committee of July 10, 1979 recommending dissolution of the Section on Cancer.

IT WAS MOVED AND CARRIED APPROVING THE RECOMMENDATION OF THE PROFESSIONAL COMMITTEE FOR DISSOLUTION OF ITS SECTION ON CANCER.

Scientific Assembly Committee

Annual Meeting Registration Fees

The Board considered the recommendation of the Scientific Assembly Committee of June 2, 1979 that $10.00 of the Association membership dues be allocated and earmarked for the scientific portion of ArMA's Annual Meeting and, following discussion,

IT WAS MOVED AND CARRIED TO REFER THE RECOMMENDATION OF THE SCIENTIFIC ASSEMBLY COMMITTEE THAT $10.00 OF ASSOCIATION MEMBERSHIP DUES BE ALLOCATED AND EARMARKED FOR ANNUAL MEETING EXPENSES AND THAT THERE BE NO FEE FOR MEMBER REGISTRATION FOR THE SCIENTIFIC PORTION OF ArMA'S ANNUAL MEETING TO THE FINANCE COMMITTEE FOR ITS REVIEW AND CONSIDERATION IN A TIMELY FASHION SO THAT A REPORT OF THE FINANCE COMMITTEE'S RECOMMENDATIONS MIGHT BE CONSIDERED BY THE BOARD OF DIRECTORS AT ITS NEXT REGULARLY SCHEDULED MEETING.

1980 Annual Meeting

The new dates and location of the 1980 Annual Meeting are April 23 - April, 1980 at The Pointe at-7677 North 16th Street, Phoenix. RECEIVED.

ArMA Auxiliary

Letter of appreciation dated 5/25/79 from Kathie DiCenso, Auxiliary Covention Chairman. RECIEVED

AMA Delegation Report

AMA Regional Meeting

Report of Arthur V. Dudley, Jr., M.D. on AMA Regional Meeting. RECEIVED

Report - 1979 Annual Meeting of AMA

Edward Sattenspiel, M.D., reported on several of the more interesting aspects at the 1979 Annual Meeting of the AMA held in Chicago, July 22- 26, 1979.

Residents' Report - AMA Annual Meeting

The joint report of William W. Brown, III, M.D. and C. Clayton Griffin, M.D. together with a brief oral report by Dr. Brown regarding the residents' impression of the AMA Annual Meeting. RECEIVED.

Correspondence

Letter dated 5/23/79 from Robert B. Hunter, M.D., Chairman of the Board of Trustees of AMA. RECEIVED.

Letter of appreciation dated 5/8/79 from Victor Zannis, M.D. RECIEVED.

Letter date 5/16/79 from the Joint Commission on Accreditation of Hospitals. RECEIVED

Other Business

AAMSE

Information brochure re: the American Association of Medical Society Executives. RECEIVED.

Board of Directors — AAMSE

Recent election of Bruce E. Robinson, Executive Director of ArMA to the Board of Directors of the American Association of Medical Society Executives. RECEIVED.

Arizona Chapter of AMSA

The Board considered the request of the Arizona chapter of the American Medical Student Association (AMSA) that a representative of their organization be an invited guest to ArMA Board of Directors' meetings, and following discussion,

IT WAS MOVED AND CARRIED THAT THE ASSOCIATION INCLUDE ON ITS ROSTER OF GUESTS TO BE INVITED TO ALL FUTURE BOARD OF DIRECTORS' MEETINGS A MEMBER OF THE ARIZONA CHAPTER OF THE AMERICAN MEDICAL STUDENT ASSOCIATION (AMSA) AND THAT SUCH ASSOCIATION BE CONTACTED WITH RESPECT TO THE NAME OF THE INDIVIDUAL THEY CHOOSE TO SO REPRESENT THEM.

Advisory Council on Nursing Manpower and Educational Needs

Dr. Linkner presented the Baord with a request for $5,000 or more from the Advisory Council on Nursing Manpower and Educational Needs, which sum would be used in the intital investigation, compilation and review of the data necessary in determing the nursing manpower in the State of Arizona.

IT WAS MOVED AND CARRIED TO REFER TO THE FINANCE COMMITTEE THE REQUEST FOR $5,000 OR MORE RECEIVED FROM THE ADVISORY COUNCIL ON NURSING MANPOWER AND EDUCATION NEEDS WITH THE REQUEST THAT THE FINANCE COMMITTEE REPORT BACK TO THE BOARD OF DIRECTORS WITH ITS RECOMMENDATION AT THE EARLIEST POSSIBLE DATE.

IRS Investigation

The Board discussed at length what appears to be an ongoing situation affecting a majority of the physicians in the State of Arizona, that is, the IRS audits or investigations pertaining to the deduction by physicians of premiums for malpractice insurance and/or capital contributions.

IT WAS MOVED AND CARRIED THAT COUNSEL FOR THE ASSOCIATION BE REQUESTED TO INVESTIGATE AND DETERMINE WHAT LEGAL ACTIONS, IF ANY, HAD BEEN FILED BY ANY OF THE PHYSICIANS IN THE STATE OF ARIZONA AGAINST THE INTERNAL REVENUE SERVICE REGARDING AUDITS OR INVESTIGATIONS PERTAINING TO DEDUCTIONS OF PREMIUMS FOR MALPRACTICE INSURANCE AND/OR CAPITAL CONTRIBUTIONS AND THAT COUNSEL FURNISH TO THE ASSOCIATION LEGAL OPION AS TO WHAT, IF ANY, ACTION SHOULD BE TAKEN BY THE ASSOCIATION ON BEHALF OF ITS MEMBERSHIP.

ArMA Annual Meeting *
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23-27, 1980 ArMA
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Quinamm™

AVAILABLE ONLY ON PRESCRIPTION

Brief Summary

INDICATIONS: For the prevention and treatment of nocturnal recumbency leg muscle cramps, including those associated with arthritis, diabetes, varicose veins, thrombophlebitis, arteriosclerosis, and static foot deformities.

CONTRAINDICATIONS: Because of the quinine content, Quinamm is contraindicated in women of childbearing potential, in pregnancy, in patients with known quinine sensitivity, and in patients with glucose-6-phosphate dehydrogenase deficiency. Hemolysis (with the potential for hemolytic anemia) has been associated with a G-6-PD deficiency in patients taking quinine.

PRECAUTIONS: Thrombocytopenic purpura may follow the administration of quinine in highly sensitive patients. Recovery will follow withdrawal of the medication.
Cinchona alkaloids, including quinine, have the potential to depress the hepatic enzyme system that synthesizes the vitamin K-dependent factors. The resulting hypoprothrombinemic effect may enhance the action of warfarin and other oral anticoagulants.

ADVERSE REACTIONS: Aminophylline may produce intestinal cramps in some instances, and quinine may produce symptoms of cinchonism, such as tinnitus, dizziness, and gastrointestinal disturbance. If ringing in the ears, deafness, skin rash, or visual disturbances occur, the drug should be discontinued.

DOSAGE AND ADMINISTRATION:
1 tablet upon retiring. When necessary, 1 additional tablet may be taken following the evening meal.

Product Information as of September, 1977

U.S. Patent 2,985,558

Merrell

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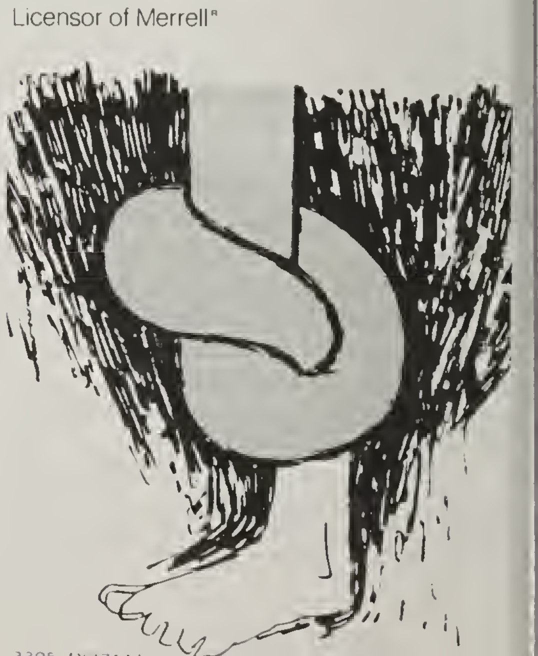

for Knotts in the night

Quinamm™

each tablet contains quinine sulfate 260 mg., aminophylline 195 mg.

specific therapy for painful night leg cramps

Nocturnal recumbency leg muscle cramping is frequently an unwelcome bedfellow for many patients—especially those with arthritis, diabetes or peripheral vascular disease . . . consider Quinamm . . . simple, convenient dosage—usually just one tablet at bedtime . . . can provide restful, welcome sleep without night leg cramps.

See opposite page for prescribing information.

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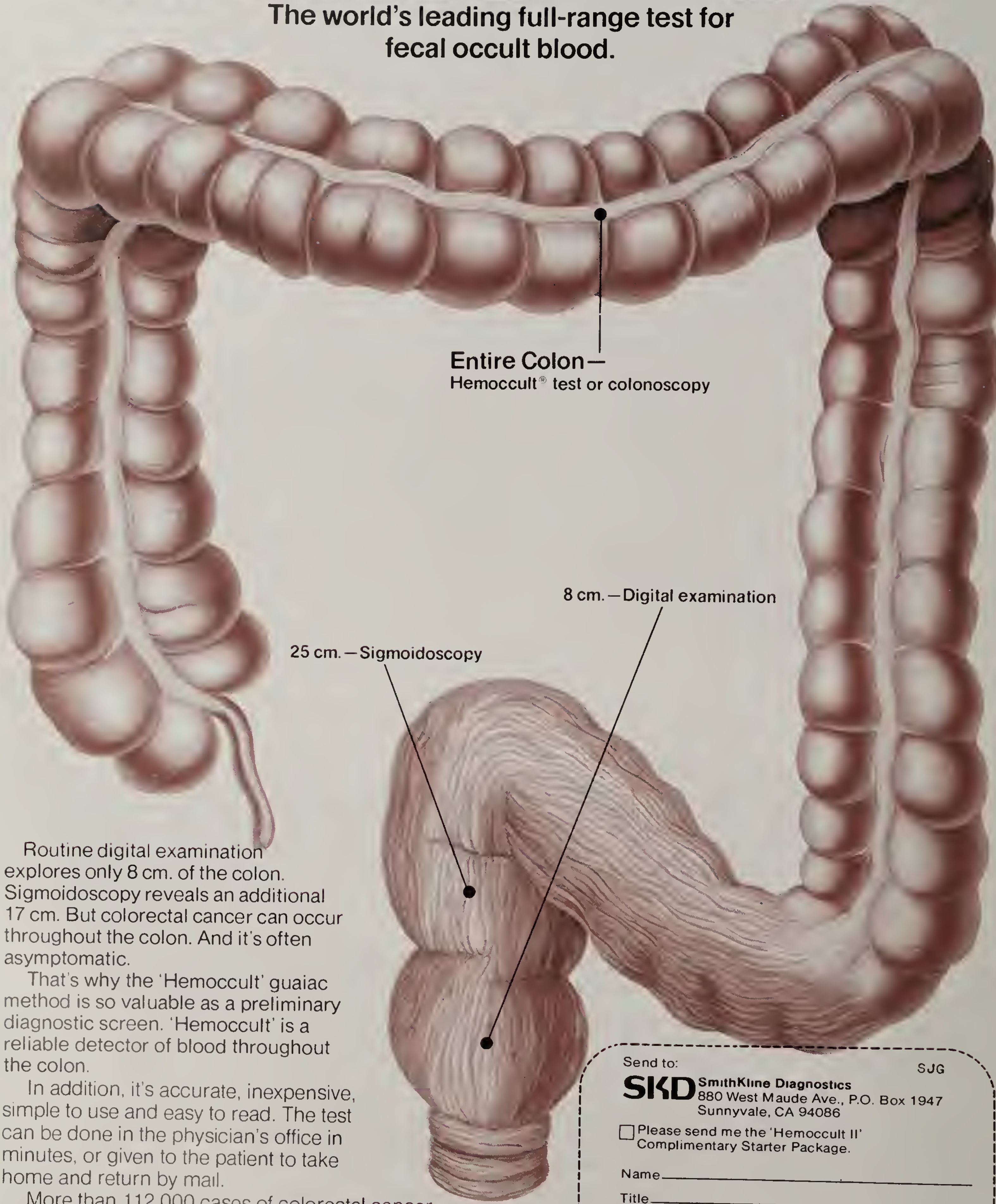
The world's leading full-range test for fecal occult blood.



Routine digital examination explores only 8 cm. of the colon. Sigmoidoscopy reveals an additional 17 cm. But colorectal cancer can occur throughout the colon. And it's often asymptomatic.

That's why the 'Hemoccult' guaiac method is so valuable as a preliminary diagnostic screen. 'Hemoccult' is a reliable detector of blood throughout the colon.

In addition, it's accurate, inexpensive, simple to use and easy to read. The test can be done in the physician's office in minutes, or given to the patient to take home and return by mail.

More than 112,000 cases of colorectal cancer will occur in the United States this year. The earlier they are diagnosed, the greater the chances for successful treatment.

'Hemoccult' is available through local distributors, nationwide.

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☐ Please send me the 'Hemoccult II' Complimentary Starter Package.

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Institution____________________

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Future Medical Meetings

The following institutions and organizations have been accredited for their continuing medical education programs by the Liaison Committee on Continuing Medical Education

- Arizona Heart Institute
- Arizona Medical Association
- Arizona State Hospital, Phoenix
- Camelback Hospital, Phoenix
- Desert Samaritan Hospital, Mesa
- Good Samaritan Hospital, Phoenix
- Health Maintenance Associates, Phoenix
- Maricopa County General Hospital, Phoenix
- Mesa Lutheran Hospital, Mesa
- Phoenix Baptist Hospital & Health Center
- Phoenix Indian Medical Center
- Phoenix Memorial Hospital
- Phoenix Psychiatric Council
- St. Joseph's Hospital & Medical Center, Phoenix
- St. Lukes's Hospital & Medical Center, Phoenix
- St.Mary's Hospital, Tucson
- Scottsdale Memorial Hospital
- Tucson Hospitals Medical Education Program, Tucson
- Veterans Administration Hospital, Phoenix
- Veterans Administration Hospital, Prescott
- University of Arizona College of Medicine, Tucson

The accredited institutions and organizations above produce a variety of continuing medical education programs. Each accredited institution and organization is responsible for designating which of these programs meet ArMA's requirements for Category 1 credit. Physicians who participate in programs which are designated Category 1 by accredited institutions will receive Category 1 credit toward the ArMA Certificate in CME and the AMA's Physician's Recognition Award.

OCTOBER

Eighth Annual Symposium of the Western Division of the American Geriatrics Society. Management of Vascular Disease in the Elderly
October 26-27, 1979. Lakes Club, Sun City, Arizona. Sponsor: Univ. of Arizona College of Medicine and Walter O. Boswell Memorial Hospital. Contact: Julius Schwimmer M.D., 13220 105th Ave., Bldg. B, Rm 61, Sun City, AZ 85351. Approved for 11 hours of category 1 credit toward ArMA, AMA & AAFP CME Certificates.

NOVEMBER

Oncology for the Non-Oncologist
November 3 & 4, 1979. Doubletree Inn, Scottsdale, AZ. Sponsor: Univ. of Arizona College of Medicine and Memorial Hospital of Phoenix. Contact: Phoenix Memorial Hospital, Medical Staff Office, 1201 S. 7th Ave., P.O. Box 21207, Phoenix, AZ 85036

Symposium on Stress
November 3 & 4, 1979. Sheraton-Place Hotel, San Francisco, CA. Sponsor: University of California Schools of Medicine, Nursing, Denistry, and Pharmacy. Contact: Univ. of California, Continuing Education Health Sciences, 24 Kirkham, San Francisco, CA 94143. CME Credit available.

Southern Medical Association Annual Scientific Assembly
November 4-7, 1979. Las Vegas, Nevada. Sponsor: Southern Medical Assoc. & AMA. Contact: AMA Dept. of Meeting Services 535 N. Dearborn St., Chicago, Ill. or Southern Medical Assoc. 2601 Highland Avenue. Biringham, Alabama 35205.

27th Annual Meeting of the Medical Society of the United States and Mexico.
November 14-17, 1979. Doubletree Inn, Tucson, AZ. Sponsor: Medical Society of the United States and Mexico. Contact: Mrs. Carolyn Parsons, Executive Secretry, 3161 N. Pantano Road, Tucson, AZ 85715.

"Wellness—An Arthritis Reality"
November 17, 1979, Sheraton-Scottsdale Inn. Sponsor: Arthritis Foundation, Central Arizona Chapter. Contact: Diona Siedler, Program Services Coordinator, Arthritis Foundation Central Arizona Chapter, 100 W. Osborn Rd, Ste D, Phoenix, AZ 85013

ACLS Provider Course
November 16-17, 1979. Arizona Medical Association offices, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Sponsor: Arizona Affiliate of the American Heart Assoc. Contact: Carol Turner, ArMA Offices, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

Arizona Society of Otolaryngology and Maxillo-Facial Surgery
November 16 & 17, 1979. The Wigwam, Litchfield Park, AZ. Sponsor: Arizona Society of Otolaryngology & Maxillo-Facial Surgery. Contact: Neil O. Ward, M.D., 4550 N. 51st. Ave., Phoenix, AZ 85031.

The Third Annual National Symposium on Aging
November 17 & 18, 1979. Holiday Inn, Golden Gateway, San Francisco, CA. Sponsor: Univ. of California San Francisco. Contact: Univ. of California, Continuing Education Health Sciences, 24 Kirkham, San Francisco, CA 94143.

DECEMBER

ECG Interpretation and Arrhythmia Management
December 7-9, 1979, Hyatt Regency, Phoenix, AZ. Sponsor: International Medical Education Corp., 64 Inverness Drive East, Englewood, Co 80112. Approved for 15 hours category 1 credit by AMA, 15 prescribed hours by AAFP, and 13 hours category 1 by ACEP.

8th Annual Seminar: Bone and Joint Diseases
December 8 and 9, 1979, Camelback Inn, Scottsdale, AZ. Sponsor: Maricopa County General Hospital. Contct: Alex Newman, M.D., Program Chairman, Dept. of Radiology, Maricopa County General Hospital, 2601 E. Roosevelt Street, Phoenix, AZ 85008. Approved for 14 hours of category 1 credit.

ACLS Provider Course
December 14 & 15, 1979. ArMA Offies, 810 W. Bethany Home Rd., Phoenix, AZ. Sponsor: Arizona Affiliate of the American Heart Assoc. Contact: Carol Turner, ArMA Offices 810 W. Bethany Home Road, Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

1980

FEBRUARY

New Perspectivies in Cerebral Localization
February 7-9, 1980. Hyatt-Regency Hotel, Phoenix, AZ. Sponsor: St. Joseph's Hospital and Medical Center, Barrows Neurological Institute. Contct: Richard A. Thompson, M.D., Chairman, 350 West Thomas Rod, Phoenix, AZ 85013.

Coronary Disease, Exercise Testing & Cardiac Rehabilitation
February 8-10, 1980. Mountain Shadows Resort, Scottsdale, AZ. Contact International Medical Education Corp., 64 Inverness Drive East, Englewood, Co 80112. Approved for 13 hours category 1 credit by AMA, and 13 prescribed hours by AAFP

5th Annual Seminar - Radiology & Geriatric Medicine
February 8-10, 1980. Doubletree Inn, Scottsdale, AZ. Sponsor: Maricopa County General Hospital. Contact: Austin R. Sandrock, M.D., Program Chairman, Maricopa County General Hospital, Dept. of Radiology, 2601 East Roosevelt Street, Phoenix, Z 85008. Approved for 15 required hours of category 1 credit.

Taos Medical Symposium
February 12-15, 1980. Tennis Ranch of Taos, Taos, New Mexico. Sponsor: New Mexico Chapter, AAFP, CME Committee, New Mexico Medical Society, Albuquerque Academy of Family Physicians, Albuquerque & Bernalillo County Medical Association. Contact: Thomas A. Bodnar, Program Director, 2650 Yale Blvd., S.E., No. 103, Albuquerque, New Mexico 87106

Second Annual International Cataract Surgery Symposium
February 18-10, 1980. Sponsor: Arizona Foundation for Ophthalmic Research and Education. Contact: Ronald W. Barnet, M.D., Medical Director, 421 N. 18th St., Ste 105, Phoenix, AZ 85006. (602) 257-1077. Approved for 12 to 22 hours of category 1 credit.

8th Annual Medicine Conference Advances In Internal Medicine II
February 21-23, 1980. Carefree Inn & Resort, Carefree Arizona 85008. Sponsor: Maricopa County General Hospital. Contact: Myra DaBolt, 2601 E. Roosevelt St., Phoenix, AZ 85008. Approved for 21 hours category 1 credit.

Gastroenterology for Clinicians - New Directions
February 21-23, 1980. Del Webb's TowneHouse, Phoenix, AZ. Sponsor: David C.H. Sun Memorial Institute. Contact: Mrs. David C.H.Sun, David C.H. Sun Memorial Institute, 4129 East Sandy Mt. Rd., Scottsdale, AZ 85253.

Pediatric Update 1980
February 25-28, 1980. Sponsor: St. Joesph's Hospital and Medical Center, Dept. of Pediatrics. Contact: Ronald A. Christensen, M.D., Dept. of Pediatric Education. St. Joseph's Hospital and Medical Center, 350 W. Thomas Rd., Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

Third Annual Mid-Winter Symposium in OB/GYN
February 27, 28, 29, 1980. Scottsdale Hilton Hotel, Scottsdale, AZ. Sponsor: Maricopa County Hospital, Dept. of OB/GYN and Phoenix OB/GYN Society. Contact: The Secretary, Phoenix, OB/GYN Society, 333 East Virginia, Ste 222, Phoenix, AZ 85004.

Contemporary Managment of Stroke and Coronary Disese
February 27 - March 1, 1980. Lodges in Taos, New Mexico. Sponosr: Symposia de Santa Fe. Contact: W.J. Levy, M.D., Symposia de Santa Fe, P.O.. Box 5175, Santa Fe, New Mexico 85702

MARCH

Durango, Colorado Ski Symposium
March 5 and March 9, 1980. Sponsor: Albuquerque Academy of Family Physicians. Conact: Albuquere Academy of Family Physicians, c/o New Mexico Medical Society, 2650 Yale Blvd., S.E., Ste 105, Albuquerque, New Mexico 87106.

Sixteenth Annual Arizona Chest Symposium
March 28-30, 1980. Doubletree Inn, Tucson, Arizona. Sponsor: University of Arizona College of Medicine, and Tucson Medical Center. Contact: Linda Alpert, R.N., Symposium Coordinator, Tucson Medical Center, P.O. Box 6067, Tucson, AZ 85733. Approved for category 1 credit.

APRIL

Emergency Room Radiology-Revised

April 11-13, 1980. Arizona Biltmore, Phoenix, Arizona. Sponsor: Maricopa County General Hospital. Contact: Austin R. Sandrock, M.D., Program Chairman, Maricopa County General Hospital, 2601 E. Roosevelt St., Phoenix, AZ 85008

MONTHLY OR WEEKLY

Shrine Medics Meeting
Second Tuesday of each month, Doctors Hospital, 5:45 p.m. J. South Classroom. Sponsor: Shrine Medics. Contact: Robert C. Briggs, M.D., 5121 N. Central Ave., Phoenix, AZ 85012.

Office Psychiatry for the Primary Provider
2nd Monday of month, 4811 N. 7th Street, Phoenix, AZ. Sponsor: Arizona Health Plan. Contact: T.R. Bittker, M.D., Box 5000, Phoenix, AZ 85010. Approved for category 1 credit.

Counter Transference Group
Weekly, Thurs. 8—10 p.m. Sponsor: Phoenix Psychiatric Council. Contact: James E. Campbell, M.D., 5051 N. 34th St., Phoenix, AZ 85018. Approved for 2 hours category 1 credit.

Monthly Meeting of Tucson Radiologists
Last Tues. of month. Plaza International, Tucson, AZ. Sponsor: U of A Medical Center, Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., U of A Health Sciences Center, Dept. of, Radiology, Tucson, AZ 85724. Approved for 2 hours category 1 credit.

Pediatric Grand Rounds
Tuesday 7:30-8:30 a.m in Phoenix: 1st Tues.—Phoenix Indian Hospital, 2nd Tues.—Maricopa County Hospital, 3rd Tues.—Good Samaritan Hospital, 4th Tues.—St. Joseph's Hospital. Sponsor: Maricopa County Hospital (Phoenix Hospital's Affiliated Pediatric Program). Contact: J. Kipp Charlton, M.D., 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 1 hour per session category 1 credit.

Review of Forensic Pathology Current Case, Special Topics
Thursday, weekly, 11 a.m., 120 S. 6th Ave., Phoenix, AZ. Sponsor: Arizona Society of Pathologists. Contact: H.H. Karnitschnig, M.D., 120 S. 6th Ave., Phoenix, AZ. Approved for 1 hour per session category 1 credit.

ARIZONA HEART INSTITUTE

4800 N. 22nd St., Phoenix, P.O. Box 10,000, Phoenix, AZ 85064. Contact: Edward B. Diethrich, M.D., Medical Dir.

Clinical Conference Cardiovascul Medicine
Third Tues., second floor classroom

ARIZONA STATE HOSPITAL

2500 E. Van Buren, Phoenix, AZ. 8500
Contact: Arnold L. Kendall, M.D.

A.S.H. Psychiatric Grand Rounds
2nd Wed., 1:00-2:00 P.M., J-6 Conf. Rm
Contact: Dr. Conger & Staff
Clinical-Pathological Conference
3rd Wed., 1:30-2:30 P.M. General Service Bldg., Conf. Rm.
Medical Grand Rounds
4th Wed., 1:00-2:00 P.M., Medical Bldg. Con Rm.
Staff Education Conference
Weekly, Wed., 1 p.m., Contact: Howard Wulsin, M.D.

BARROW NEUROLOGICAL INSTITUTE

Medical Education
Barrow Neurological Institute of St. Joseph Hospital and Medical Center, 350 W. Thoma Rd., Phoenix, AZ 85013. Sponsor: St Joseph' Hospital & Medical Center. Contact: John Green, M.D. Approved for 1 hour category credit.

Neurology Teaching Conference
Tuesdays, 8:30-9:30 a.m., Eighth Floor Con Room.

Neurosurgery Morbidity Conference
Wednesdays, 8:15-9:15 a.m., on first and thir weeks, Eighth Floor Conference Room.

Neuro-ophthalmology Conference
Tuesdays, 7:30 a.m. in 8th floor neurolog conference room.

Spinal Injury Conference
Wednesdays, 8:15-9:15 a.m., on second an fourth weeks, in Neuropathology Con Rm.—a multidisciplinary review of admissio by neurosurgeons, orthopedists, an rehabilitation specialists

Neuropathology of Gross Specimen Conference
Thursday, 7:30-8:30 a.m. in the Morgue.

Neurology-Neurosurgical Clinical-Neuroradiological and Neuropathological Conference
Fridays, 8-9 a.m., First Floor Conference Rm

Neuropathology or Neuroradiolog Conferences
Friday, 9 a.m., Neuropathology i Neuropathology Conference Rm. Neuroradiology in First Floor Conferenc Rm.

Neurorehabilitation Conference
Tuesdays, noon, 8th Floor Conference Rm.

Neurosurgical Journal Club
Saturdays, 9-11 a.m. in Eighth Floo Conference Rm.

An Invitation from....

Robert Chahine, M.D., F.A.C.C.
Associate Professor of Medicine, Baylor College of Medicine

Robert Schnitzler, M.D., F.A.C.C.
Director, Intensive Care Units, Bexar County Hospital, San Antonio

John Schroeder, M.D., F.A.C.C.
Assistant Professor of Medicine and Cardiology, Stanford University

Basil Williams, M.D., F.A.C.C.
Associate Clinical Professor of Medicine, University of Utah College of Medicine

Frank Yanowitz, M.D., F.A.C.C.
Assistant Professor of Medicine, University of Utah College of Medicine

...to meet with them in the highly acclaimed postgraduate seminar:

ECG Interpretation and Arrhythmia Management

December 7-9, 1979
Hyatt Regency, Phoenix, AZ

Each of these distinguished faculty members is nationally recognized for his expertise and communicative ability. You won't attend a more informative program on this timely and important topic, and we hope that you accept our invitation.

Credit: 15 hours A.M.A. Category I; 15 prescribed hours of A.A.F.P.; 13 hour of A.C.E.P. Category I

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Other CME Seminars:
- ☐ Coronary Disease, Exercise Testing and Cardiac Rehabilitation
- ☐ Cardiac Ischemia and Arrhythmia-Current Concepts for Diagnosis and Treatment
- ☐ EKG Interpretation and Arrhythmia Management
- ☐ Cardiac Rehabilitation

CAMELBACK HOSPITAL

5055 N. 34th St., Phoenix, AZ 85018. Sponsor: Camelback Hospital. Contact: Medical Director.

Patient Staffing Conference
Three times weekly.

DESERT SAMARITAN HOSPITAL

1400 South Dobson Road, Mesa, Arizona, Contact: L.A. Rosati, M.D. Approved for category 1 credit.

Clinical Conference - Dept. of Medicine
Weekly Thurs., 12:30-1:30 P.M. Buffet Lunch, Grande Rm.
Medical Education Programs
Wed.,7:00 P.M. Desert Samaritan Hospital, Mesa.

Monthly Visiting Professor Programs
Tuesday 7:00 P.M., Buffett 6:00 P.M.
OB/GYN Medical Staff Conference
Weekly, Monday, 12:30-1:30 P.M. Grande Rm.

Surgical Morbidity & Mortality Conference
2nd Thurs., 7:00 A.M., Grande Rm.

GOOD SAMARITAN HOSPITAL

1033 East McDowell Rd., Phoenix, AZ Contact: Warren A. Hinchcliffe, M.D. Approved for category 1 credit.

Anatomical Path. & Cytology
Weekly, Mondays 4:00-5:00 P.M., S-4 Conf Rm.
Gynecological Section Conference
2nd Monday, 12:30-1:30 P.M., West Rm
Monday Noon Conference
Weekly, 12:00-1:00 P.M. R-2 Conf. Rm.
Mortality/Morbidity, Surgery Comm.
2nd Monday, 11:30-2:00 P.M., West Rm.
Obstetrical Section Conference
1st Monday, 12:30-1:30 P.M., West Rm.
Pulmonary Disese Grand Rounds
Weekly, 12:00-1:00 P.M., D-5 North
Family Practice Conference
Weekly Tuesday, 12:00-1:00 P.M., Fam. Practice Conf. Rm.
Pediatric Grand Rounds
3rd. Tuesday, 7:300-8:30 A.M., West Rm.
Peri-Natal Mortality Conference
3rd Tuesday, West Rm. (May-August).
Surgical Grand Rounds
Weekly, Tuesday 7:00-8:30 A.M. East Rm.
Clinical Cancer Fourm
3rd Wednesday, 12:00-1:00 P.M., West Rm, Contact: John A. Bruner, M.D.
Combined Cardiovascular & Family Practice Conf.
Ist, 2nd, & 4th Wed., 12:00-1:00 P.M., D-5 North.
Dr. Finger's Lecture
Weekly, Wed., 12:30-1:30 P.M., R-2 Conf. Rm.
Learning Disabilities
Weekly, 10:30-12:30 A.M., R-2 Conf. Rm.
OBGYN Dept. Review & Statistics
Weekly, 12:30-3:00 P.M., OBGYN Conf. Rm.
PM&R Grand Rounds
1st. & 3rd. Wed., 12:00-1:00 P.M., R-2 Conf. Rm.
Gynecological Pathology
Weekly Thursday, 1:00-2:00 P.M., S-4 Conf. Rm.
GI Radiology Conference
1st Thursday, 12:00-1:00 P.M., F151, Radiology Conf. Rm.
Neurology Grand Rounds
Weekly Thursday, 9:00-10:00 A.M., D-5 North.
Tumor Treatment Planning Conf.
2nd & 4th Thursdays, 12:00-1:00 P.M., F151 Radiology
Joint Tumor Gyn Conference
2nd Friday, 12:30-1:30 P.M., West Rm.
Kinesology
Weekly Friday, 4:00-5:00 P.M., R-2 Conf Rm.
Medical Grand Rounds
Weekly Friday, 8:00-9:00 A.M., West Rm.
Neuropathy Conference
Last Firday, 1:15-2:15 P.M., S-4 Morgue.
Psychiatry Grand Rounds
Weekly Friday, 11:30-1:00 P.M. , East Rm.
Pediatric Wednesday Seminar
July, Sept., March 12:30-1:30 P.M., East RM.
Pediatric Thursday Seminar
May, June, July, 12:30-1:30 P.M., East Rm.
Phoenix Ultrasound Society Meeting
Sept., thru May,2nd Thursday, 7-9:00 P.M., Med. Ed. Bldg. Contact: Dr. A. Ben-Ora.
PM&R Journal Club
Alt. Fridays, 12:00-1:00 P.M., R-2 Conf. Rm.
Family Practice Grand Rounds
1st. Wed.: April, Aug. & Dec., 7:30-9:00 A.M., West Rm.

KINO COMMUNITY HOSPITAL

2800 E. Ajo Way, Tucson, AZ 85713. Contact: Eric G. Ramsay, M.D. Approved for category 1 credit.

Attending's Hour
5th Monday, 12:30 P.M., Contact: Joseph Goldberger, M.D.
Infectious Disese Conference
3rd Monday, 12:30 P.M., Contact: Larry Lincoln, M.D.
Nephrology Conference
1st Monday, 12:30 P.M., contact: Alan Cohn, M.D.
OB/GYN Dept. Meeting
Weekly Mondays, 7:30 A.M., Contact: Lewis Shenker, M.D.
Psychiatric Conference
2nd & 4th Mondays, Contact: Jose Santiago, M.D.
Surgical Conference
Weekly Mondays, Contact: Ronald Fischer, M.D.
Cardiology Conference
4th Tuesday, 12:30 P.M., Contact: Brendan Phibbs, M.D.
Gynecology Conference
5th Tuesday, 12:30 P.M., Contact Lewis Shenker, M.D.
Neurology Conference
1st & 3rd. Tuesday, 12:30 P.M., Contact: H.W. Buschsbaum, M.D.
Radiology Conference
2nd Tuesday, 12:30 P.M., Contact: Marvin Baker, M.D.
Clinical Pharmacology
4th Wed., 12:30 P.M., Contact: Lee Gardner Pharm.D.
Clinical Pathology Conf.
2nd Wed., 12:30 P.M., Contact: Philip Altman M.D.
Gynecology Pathology Conference
Weekly Wed., 7:3'45 A.M., Contact: Lewis Shenker, M.D.
Medical Conference-Mortality & Morbidity
3rd Wed., 12:30 P.M., Contact: Joseph Goldberger, M.D.
Rheumatology Conference
1st Wed. 12;30 P.M., Contact: George Werner M.D.
Cardiology Conference
1st Thursday, 12:30 P.M., Contact: Brendan Phibbs, M.D.
Clinical Correlation
3rd Thursday, 12:30 P.M., Contact: Elliot Munjack, M.D.
G-I Conference
4th Thursday, Contact: Ray Moldow, M.D.
OB/GYN Sonography/X-ray Conference
Weekly Thursday, 9:15 A.M., Contact: Lewis Shenker, M.D.
Chest Conference
2nd & 4th Fridays, 12:30 P.M., Contact: David Engelsberg, M.D.
Dermatology Conference
3rd Friday, 12:30 P.M., Contact: Peter Lynch, M.D
Hematology Conference
1st Friday, 12:30 P.M., Contact: Steven Ketchel, M.D.

MARYVALE SAMARITAN HOSPITAL

Continuing Medical Education Program
Every other Wednesday, 7-8 a.m.

MARICOPA COUNTY GENERAL HOSPITAL

2601 E. Roosevelt, Phoenix, Az 85008. Contact: Steven Schaffner, M.D.

Medicine Conference
Daily 12:00-1:00 P.M., Contact: Steven Schaffner, M.D.
Chest Surgery Conference
Weekly Mondays, 1:30 P.M., Satan Cruz Rm.
Obstetrical Problem Conference
Weekly Tuesday, 7:15 A.M., Yavapai Rm
Orthopedic Conference
Weekly Tuesday, 7:30 A.M., Santa Cruz Rm.
Pediatric Grand Rounds
2nd Tuesday, 7:30-8:30 A.M.
Hand Surgery Conference
Weekly Wed., 7:30 A.M., Santa Cruz Rm.
Neurosurgery Discharge Planning
Weekly Wed., 1:30 P.M., Station 42.
OB/Neonatology Seminar
Weekly Wed. 7:15 A.M., Yavapai Room
Psychiatry Conference
Weekly Wed. 11:00-12:00 A.M.
Surgery Conference
Weekly Wed., 7:00-8:00 A.M.
Cardiac/Cardiology Conference
Weekly Thurs. 12:00 P.M., Yavapai Room.
OB/GYN Resident Conference
Weekly Thurs. 12:00 P.M., Yavapai Rm.

GYN, Endocrine Seminar
Weekly Friday, 12:30 P.M., Santa Cruz Rm.
GYN Grand Rounds
Weekly Friday, 1:30 P.M., Santa Cruz Rm.
OB/GYN Surgical Pathology Conf.
Weekly Friday, 7:00 A.M., Yavapai Rm.
Orthopedic X-Ray Conference
Weekly Friday, 7:30 A.M., Santa Cruz Rm.

PHOENIX BAPTIST HOSPITAL & MED. CENTER

6025 N. 20th Ave., Phoenix, AZ 85015. Contact: James L. Grobe, M.D. Approved for category 1 credit.

Medical & Surgical Conference
Weekly Monday, 12;00 Noon
Medical Conference
1st. & 3rd Tuesdays, 12:00 Noon.
Medical & Surgical Conference
Weekly Wed., 12:00 Noon.
Medical & Surgical Conference
Weekly Friday, 12:00 Noon.

PHOENIX INDIAN MEDICAL CENTER

4212 North 16th St. Phoenix, AZ 85016. Contact: Leland L. Fairbanks, M.D. Approved for category I credit.

Pediatric Grand Rounds
1st Tuesday, 7:30-8:30 A.M.
Clinical Staff Teaching conference (Family Practice)
Weekly Wed., 8:00-9:00 A.M., Conf. Rm. A.

PHOENIX MEMORIAL HOSPITAL

1201 S.7th Ave. Phoenix, AZ. 85036. Contact: George Scharf, M.D., Approved for Category 1 credit.

Monthly Medical Education Seminar
3rd Monday, 6:30 P.M., Kiva Conf. Rm.
Tumor Board Conference
Weekly Friday, 12:00 P.M., Kiva Rm., Contact: Howard Kimball, M.D.

SCOTTSDALE MEMORIAL HOSPITAL

7300 East 4th St., Scottsdale, AZ 85251. Contact: W. S. Williams, M.D. Approved for category 1 credit.

Family Practice Conference
1st Monday, 12:45 P.M.
Morbidity & Mortality Conference
2nd Monday, 12:45 P.M.
Medical Grand Rounds
3rd Monday, 12:45 P.M.
Neurology & Neurosurgery Conference
3rd Monday, 12:30 P.M., Doctors' Lounge
Internal Medicine-Clinical Pathlogical Conference
4th Monday, 12:45 P.M.
Pulmonary Medicine Conference
1st Tuesday, 12:30 P.M., Doctors' Lounge
Cardiology Conference
2nd Tuesday 12:30 P.M., Doctors' Lounge
Surgery Departmental Meeting
3rd Tuesday, 12:15 P.M., Doctors' Lounge
Thoracic & Vascular Surgery Conference
4th Tuesday, 12:30 P.M., Doctors' Lounge
Cardiopulmonary Conference (Didactic)
4th Tuesday, 12:30 P.M., Doctors' Lounge
Peri-Natal Mortality & Morbidity Conf.
2nd Wed., 12:30 P.M., Doctors' Lounge
Trauma Conference
3rd Thurs., 12:30 P.M., Doctors' Lounge
Tumor conference
Last Thursday, 12:30 P.M., Doctors' Lounge
Psychiatry & Behavioral Sciences Conference
1st Friday, 12:30 P.M., Doctors' Lounge
GI Medical-Surgical-Radiological Conference
2nd Friday, 12:30 P.M., Doctors' Lounge
Uronephrology-Oncology
Last Friday, 12:30 P.M., Doctors' Lounge

ST. JOSEPH'S HOSPITAL PHOENIX

350 West Thomas Road, Phoenix, AZ 85013. Contact: Joseph C. White, M.D.

OB/GYN Section Conference
3rd & 4th Mondays, 12:30-1:30 P.M., 1st Floor Conf. Rm.
Pediatric Grand Rounds
4th Tuesday, 7:30-8:30 A.M., Contact: J. Kipp Charlton, M.D.

ST MARY'S HOSPITAL & HEALTH CENTER

1601 W. St. Mary's Road, Tucson, AZ 85703. Contact: see below.

Monthly Specialty Conference-Dept. of Surgery
1st Monday, 7:30 A.M., Century Rm. A. Contact: Med. Staff Office.
Grand Rounds: Medical Surgical, Family Practice, Pathology, Radiology
Weekly Thursday
Cardiology Conference
Weekly Friday, 8:00-9:00 A.M., Century Rm. Contact: Anthony Forte, M.D.

U OF A HEALTH SCIENCES CENTER

Sponsor: U of A College of Medicine, Tucson, AZ 85724. Contact: See below. Approved for category 1 credit.

Anesthesiology Lectures
Weekly Thursday, 4:00-5:00 P.M.
Cardiac Catheterization Conference
Weekly Friday, 4:00 P.M., Contact: Dr. Temkin.
Cardiology Research Conference
Weekly Tuesday, 7:30 A.M., Contact: Dr. Roeske.
Case Discussion
Weekly Wed., 7:00 A.M., Din. Rm.C&D., Contact: Drs. Vaughan & Doyle..
Clinical Immunology, Allergy & Rheumatology Rounds
Every Friday, Noon-1 p.m. Contact: John Boyer, M.D., Dept. of Internal Medicine.
Cerebrovascular Disease Conference
Mondays, 5-6 p.m., weekly, Rm. 5505. Contact: Wesly Moore, M.D.,Depts. of Surgery, Nurology & Radiology.
Dean's Clinical Rounds
Contact: Louis J. Kettel, M.D.
Dermatology Rounds
Weekly Wed., 11:00 A.M., Contact: Dr. Lynch
Ear, Nose & Throat
Alternate Wed., 4:00 P.M., Contact: Dr. Coulthard
Educaton Bloc
Weekly Thurs, 4:00-5:00 P.M., Rm 5403
Endocrinology Seminar
Weekly Thursday, 12:00-1:00 P.M., Contact: Dr. Nugent
Gastrointestinal Path. Conference
4th Friday, 1:30 P.M.
Head & Neck Tumor Meeting
Alternate Wed., 4:00 P.M., Contact: Dr. Manning
Hematology-Oncology Clinical Conference
Every Tuesday, Noon-1 p.m., 1st, 3rd & 5th Tuesdays, Rm 6505, Tucson Medical Center. 2nd & 4th Tuesdays, Rm. N318, VA Hospital. Contact: Sidney Salmon, M.D., Dept. of Internal Medicine.
Joint Thyroid Clinic Conference
1st & 3rd Mondays of every month. 1 to 2 p.m., AHSC,Rm. 6505. Contact: Sidney Werner, M.D., Dept. of Internal Medicine, Endocrinology Section.
Nuclear Medicine
Thursday, 7:30 a.m., Radiology Conf. Rm., UAHSC. Contact: Irwin M. Freundlich, M.D., Dept. of Diagnostic Radiology.

Neurology Grand Rounds
Every Friday, Noon, 1st, 3rd, & 5th Fridays, UAHSC, Rm. 7505. 2nd & 4th Fridays, VA Hospital. Contact: William A. Sibley, M.D., Dept. of Neurology.
Neuromuscular Disese Conference
Weekly Friday, 11:00 A.M., Contact: Dr. Stern
Neuropathology Review Conference
Thursdays, 8:30 a.m., Rm. 5120, UAHSC. Contact: Peter C. Johnson, M.D.
Neuroradiology Conference
Weekly Thurs., 5:00 P.M., Contact: Dr. P.C. Christenson
Neurosciences Seminar
Every Friday, 7:30 a.m.,UAHSC, Rm 7505. Contact: Colin R. Bamford, M.D., or Jose F. Laguna, M.D., Dept. of Neurology.
Neuromus Journal Conference
2nd & 4th Thursday, 7:00-9:00 P.M., Contact: Dr. Stern.
Orthopedic Rounds
Saturday, 8:00 A.M., Contact: Dr. Peltier.
Pathology Seminar
Friday, 4:30-5:30 p.m., Rm. 5120, UAHSC. Contact: Paul Finley, M.D., Dept. of Pathology.
Pediatric Problem Patient Conference
Weekly Wed., 8:00 A.M., Contact Dr. Walson
Pediatric Research Forum
Weekly Tuesday, 7:30 A.M., Contact: Dr. James Jones
Pediatric Specialty Conference
Weekly Friday, 8:00 A.M., Contact: Dr. Philip Walson.

Psychiatric Grand Rounds
Weekly Wed., 12:00 Noon, Contact: Dr. Robert Cutts

(Psych.) Monthly Case Conference
2nd Friday, 7:30-9:00 A.M., Contact: Dr. Alan Levens

Pulmonary Rounds
Weekly Friday, 11:30 A.M., Contact Dr. Benjamin Burrowns.

Chest Radiology
Monday, 5-6 p.m., weekly, Rm 1535F, UAHSC. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Gastrointestinal Radiology
Mondays, 7:30 a.m., weekly except for summer months. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Radiation Oncology Planning Conference
Friday, weekly, 8:30-10 a.m., UAHSC, Rm. 0655. Contact: Robert S. Heusinkveld, M.D., Radiation Oncology Section.

Radiology-Rheumatology Conference
Thursday, 8 a.m., UAHSC, Library Rm.1535C. Contact: Irwin M. Freundlich, M.D. Dept. of Radiology.

Renal Pathology Conference
1st & 3rd Thurs., 11:30 A.M., Contact: Dr. Nagle.

Resident's Conference
Wednesday, weekly 5-6 p.m., Diag. Radiology Conf. Rm. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology

Uroradiology
Alternate Thurs, 5:00 P.M., Contact I.M. Freundlich

Surgical Literature/Basic Skills
Saturday, 8:00 A.M., Contact: Dr. P. Whitney

Surgical Morbidity & Mortality Conference
Weekly Wed., 8:00 A.M., Contact: Dr. Wangensteen.

Trauma Conference
Every Thursday,4-5 p.m. UAHSC, Rm. 5505. Contact: Martin Silverstein, M.D., Dept. of Surgery.

Grand Ward Rounds—Trauma
Every Tuesday, 8 a.m., UAHSC. Contact: Martin Silverstein, Surgery Dept., Trauma

Toxicology Conference
Weekly Tuesday, 8:00 A.M., Contact: Dr. Keith Likes
Section.

Monthly Meeting Tucson Ultrasonography Group
First Wed. of month, 4:30-5:30 p.m., UAHSC. Contact: Irwin M. Freundlich, M.D.

General Urology Conference
Weekly Thursday, 12:00 Noon, Contact: Dr. G. W. Drach.

VETERANS ADMINISTRATION HOSPITAL

7th St. and Indian School Rd., Phoenix, AZ 85012. Sponsor: VA Hospital. Contact: Alfred Heilbrumn, M.D. Approved for Category 1 credit.

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Phoenix, AZ 85036
258-5111, Ext. 331

Cancer Symposium
Weekly Wed., 1:00 P.M., Contact Dr. Berkley, Ext. 419

Cardiology Conference
2nd Thursday — 1 p.m. — T5, Dr. Habibzadeh, Ext. 348.

Clinicopathology Conference
4th Thursday, March, June, Sept. & Dec., 1:00 P.M., Contact Dr. Likos, Ext. 623

Dermatology Conference
1st, 2nd, & 3rd Wednesday (usually, but is subject to change) 10 a.m. — C118, Dr. Roberts, Ext. 426.

G.I. Clinic
2nd & 4th Wed, 1:00-4:00 P.M.,

G.I. Radiology Clinical Correlation Conference 1st & 3rd Monday — 1 p.m. — C435, Dr. Sanowski, Ext. 618.

G.I. Grand Rounds
Weekly, Wed., 8:00 A.M., G-I Ward , Contact: Dr. Severino

G.I. Resident Conference
Weekly Friday, 1:00 P.M., D446, Contacts: Drs. Sanowski & Kozarek

G.I. Medical Surgical Conference
Weekly Friday, 2:00 P.M., Path. Conf. Rm.

G.I. Rounds
Weekly Thursday, 1:00 P.M., D446

Medical Service Grand Rounds
Weekly Friday — 11 a.m. — T5, Dr. Zeller, Ext 272

Medical-Surgical G.I. Conference
1st & 3rd Friday, 2:00 P.M., Rm. 2128, Contact: Drs. Kozarek and Mertz, Ext 493

Medical-Surgical Chest Conference
1st and 3rd Thursday — 1 p.m. — T5, Dr Chowdhury, Ext. 388.

Surgery-Pathology Conference
Weekly Thursday, 2 p.m. — 2128, Drs. Mertz Ext. 493 & Likos, Ext 623

Surgery Grand Rounds
Weekly Thursday, 3:00 P.M., Contact: Dr Mertz, Ext. 493.

TUCSON VETERANS ADMINISTRATION HOSPITAL

3601 South Sixty Ave., Tucson, AZ 85723. Contacts listed below. Aprroved for category 1 credit.

Medical Surgical Chest Conference
Weekly Tuesday, 2:00 P.M., Contact: Dr. Young

Medical Grand Rounds
Weekly Wed., 12:00-1:00 P.M., V.A. Hospital Staff Conf. Rm. & (AHSC), Contact: Jay Smith, M.D.

Surgical Grand Rounds
Weekly Wed., 4:00 P.M., Contact: Dr. Putnam

Endocrinology Seminar
1st.,3rd, & 5th Thurdsays, 12:00-1:00 P.M., Rm. N318, Contact: Dept. of Internal Medicine.

Vascular Radiology, Interesting Case Conf.
Weekly Thursday, 12:00 Noon

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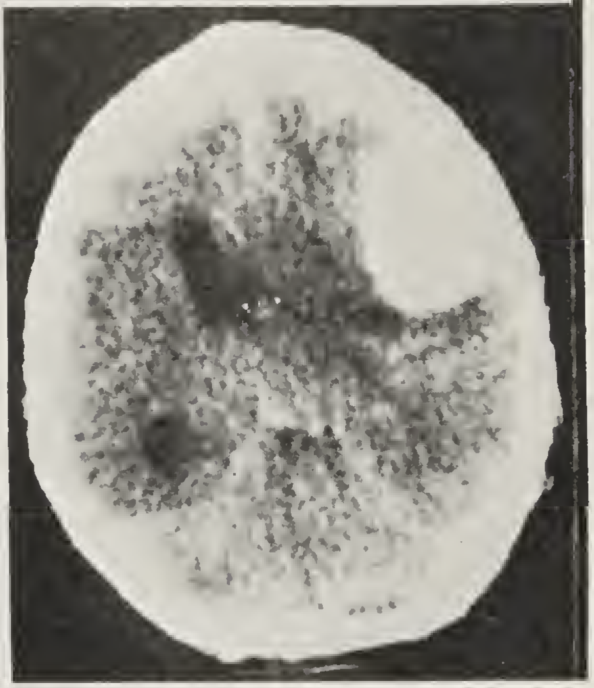

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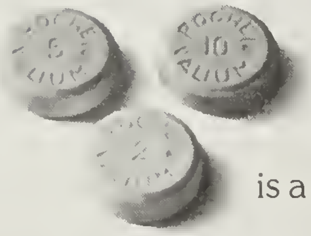

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Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

***Usage in Pregnancy:* Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated. (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® (diazepam) Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10.

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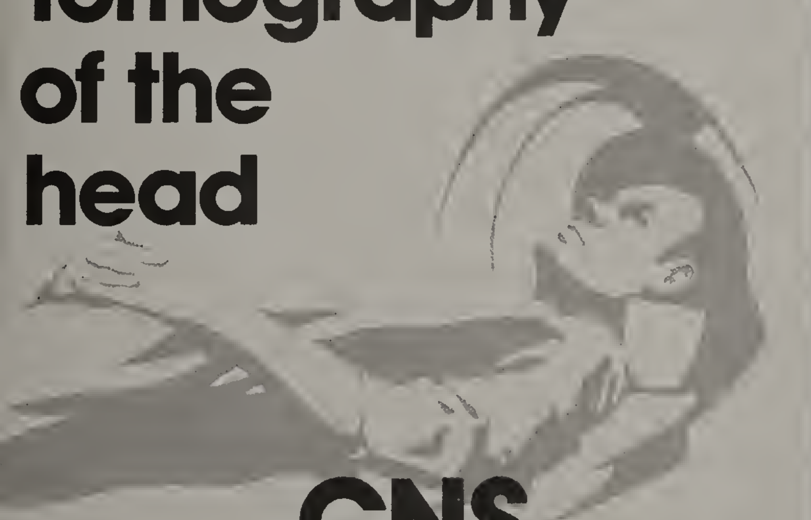

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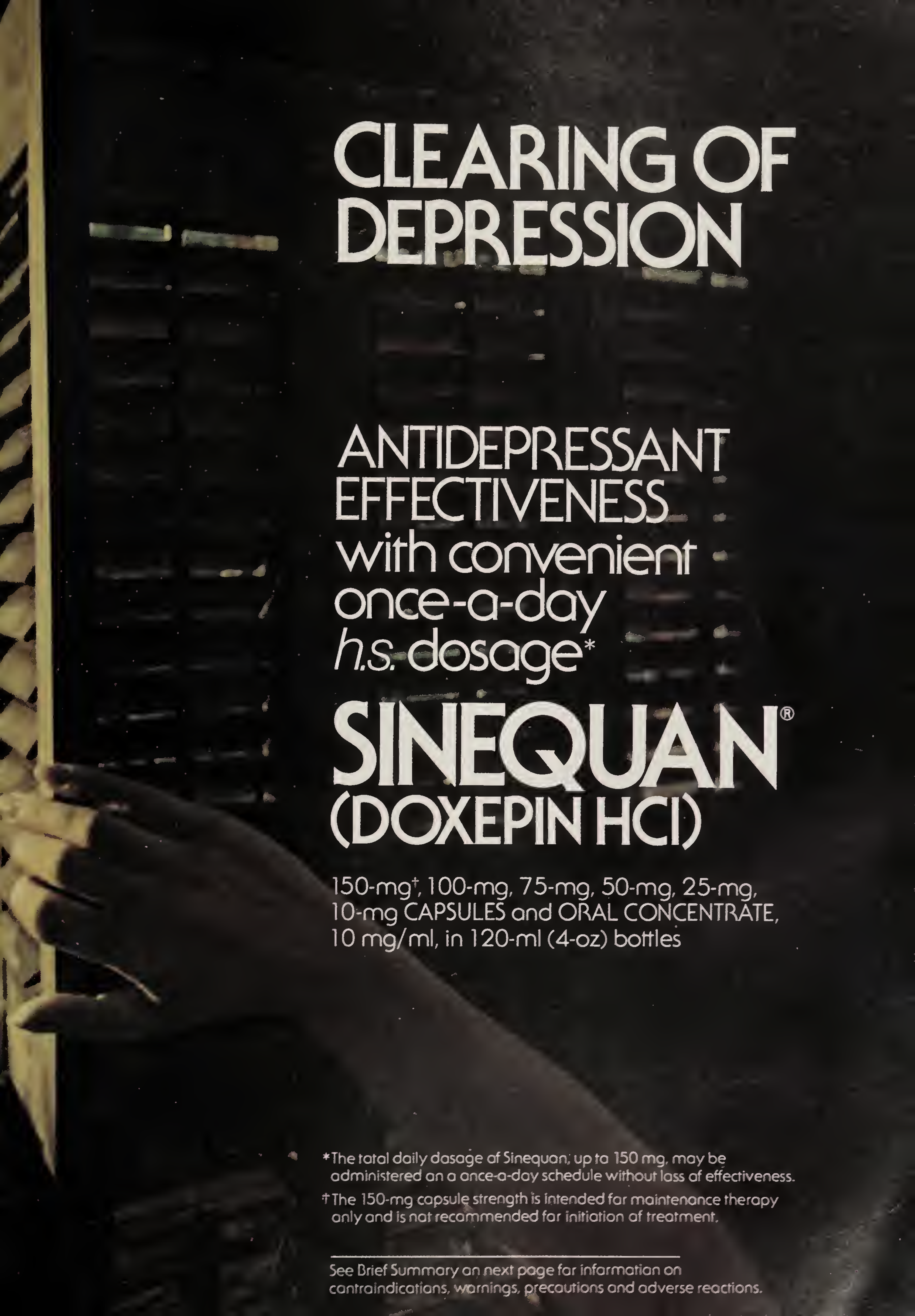

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†The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

ANTIDEPRESSANT EFFECTIVENESS

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CONVENIENT ONCE-A-DAY *h.s.* DOSAGE

which may improve patient compliance. The total daily dosage, up to 150 mg per day, may be given on a once-a-day schedule without loss of effectiveness. Sinequan may also be given on a divided dosage schedule, up to 300 mg per day.

PROMINENT SEDATIVE EFFECT

which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

USUALLY WELL TOLERATED

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have been reported occasionally. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

EXTENDED RANGE OF DOSAGE STRENGTHS

for flexibility in individualizing therapy.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients Possibility of cross sensitivity with other dibenzoxepines should be kept in mind

Warnings. The once-a-day dosage regimen of SINEQUAN (doxepin HCl) in patients with intercurrent illness or patients taking other medications should be carefully adjusted This is especially important in patients receiving other medications with anticholinergic effects.

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage This is especially important in patients who may use alcohol excessively

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug

Patients should also be cautioned that their response to alcohol may be potentiated

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy Prescriptions should be written for the smallest feasible amount

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen

Adverse Reactions. NOTE Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use However, due to the close pharmacological similarities among the tricyclics, the reactions should be considered when prescribing SINEQUAN

Anticholinergic Effects Dry mouth, blurred vision, constipation, and urinary retention have been reported If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage

Central Nervous System Effects Drowsiness is the most commonly noticed side effect This tends to disappear as therapy is continued Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures.

Cardiovascular Cardiovascular effects including hypotension and tachycardia have been reported occasionally

Allergic Skin rash, edema, photosensitization, and pruritus have occasionally occurred

Hematologic Eosinophilia has been reported in a few patients There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura

Gastrointestinal Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported (See anticholinergic effects)

Endocrine Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting daily dose of 75 mg is recommended Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg/day to 150 mg/day

In more severely ill patients higher doses may be required with subsequent gradual increase to 300 mg/day if necessary Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg/day.

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice. Some of these patients have been controlled on doses as low as 25-50 mg/day

The total daily dosage of SINEQUAN (doxepin HCl) may be given on a divided or once-a-day dosage schedule. If the once-a-day schedule is employed the maximum recommended dose is 150 mg/day This dose may be given at bedtime **The 150 mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.**

Antianxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks

Overdosage.
A Signs and Symptoms
1 Mild Drowsiness, stupor, blurred vision, excessive dryness of mouth
2 Severe: Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias and tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus), hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes
B Management and Treatment
1. Mild Observation and supportive therapy is all that is usually necessary
2 Severe Medical management of severe SINEQUAN overdosage consists of aggressive supportive therapy If the patient is conscious, gastric lavage, with appropriate precautions to prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbed The use of activated charcoal has been recommended, as has been continuous gastric lavage with saline for 24 hours or more An adequate airway should be established in comatose patients and assisted ventilation used if necessary EKG monitoring may be required for several days, since relapse after apparent recovery has been reported Arrhythmias should be treated with the appropriate antiarrhythmic agent. It has been reported that many of the cardiovascular and CNS symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intravenous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine is rapidly metabolized, the dosage should be repeated as required Convulsions may respond to standard anticonvulsant therapy, however, barbiturates may potentiate any respiratory depression Dialysis and forced diuresis generally are not of value in the management of overdosage due to high tissue and protein binding of SINEQUAN

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to: 10 mg, 75 mg, and 100 mg doxepin bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's). 25 mg and 50 mg doxepin: bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's). 150 mg doxepin: bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN Oral Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibrated at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg Each ml contains doxepin HCl equivalent to 10 mg doxepin Just prior to administration, SINEQUAN Oral Concentrate should be diluted with approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune or pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a number of carbonated beverages For those patients requiring antidepressant therapy who are on methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mixed together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water; but not with grape juice Preparation and storage of bulk dilutions is not recommended

More detailed professional information available on request.

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—Dr. William Felts, Past President,
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More and more physicians today are beginning to realize the extent of the economic influence they have, and are finding ways of holding costs down.

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More physicians today realize what a tough problem we’re all faced with. They know this is a challenge for medicine. And that physicians are in the best position to deal with and solve the problem.

**PATIENT CARE Magazine—Outlook 1977, "Face-Off: Cost Containment vs. Chaos," January 1, 1977.*

Lyle CB, et al. "Practice habits in a group of eight internists," ANNALS OF INTERNAL MEDICINE 84 (May 1976), 594-601.

Schroeder SA, et al. "Use of laboratory tests and pharmaceuticals: variation among physicians and effect of cost audit on subsequent use," JOURNAL OF THE AMERICAN MEDICAL ASSOCIATION 225 (Aug. 20, 1973), 969-73.

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The Upjohn Company announces a new indication for Motrin® (ibuprofen)

A well-tolerated, nonnarcotic prescription for pain

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Motrin now proved an effective analgesic for mild to moderate pain

Motrin 400 mg provided greater relief of pain than did propoxyphene 65 mg in controlled clinical pain studies.

Time after drug administration (hour)		.5	1	2	3	4
Mean relief-of-pain scores* (No. patients reporting)	Motrin 400 mg ibuprofen	.89 (108)	1.25 (108)	1.36 (108)	1.28 (107)	1.19 (106)
	Darvon 65 mg propoxyphene	.66 (100)	.99 (99)	1.13 (96)	.99 (96)	.80 (96)
Statistical significance		$p<0.02$	$p<0.01$	$p<0.05$	$p<0.02$	$p<0.002$

*0 = No relief 1 = Partial relief 2 = Complete relief

Data on file at The Upjohn Company

Motrin demonstrated statistically significant greater relief of pain than did Darvon at all time intervals.

Motrin 400 mg TABLETS

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- Not a narcotic • Not addictive • Not habit forming
- Rapid analgesic action • Indicated in acute and chronic pain
- Well tolerated. The most common side effect with Motrin is mild gastrointestinal disturbance.

Please turn the page for a brief summary of prescribing information.

Upjohn

Motrin® (ibuprofen) now proved an effective analgesic for mild to moderate pain

Motrin® Tablets (ibuprofen, Upjohn)

Indications and Usage: Treatment of signs and symptoms of rheumatoid arthritis and osteoarthritis during acute flares and in long-term management. Safety and efficacy have not been established in Functional Class IV rheumatoid arthritis.

Relief of mild to moderate pain.

Contraindications: Individuals hypersensitive to it, or with the syndrome of nasal polyps, angioedema and bronchospastic reactivity to aspirin or other nonsteroidal anti-inflammatory agents (see WARNINGS).

Warnings: Anaphylactoid reactions have occurred in patients with aspirin hypersensitivity (see CONTRAINDICATIONS).

Peptic ulceration and gastrointestinal bleeding, sometimes severe, have been reported. Ulceration, perforation, and bleeding may end fatally. An association has not been established. Motrin should be given under close supervision to patients with a history of upper gastrointestinal tract disease, only after consulting ADVERSE REACTIONS.

In patients with active peptic ulcer and active rheumatoid arthritis, nonulcerogenic drugs, such as gold, should be tried. If Motrin must be given, the patient should be under close supervision for signs of ulcer perforation or gastrointestinal bleeding.

Precautions: Blurred and/or diminished vision, scotomata, and/or changes in color vision have been reported. If these develop, discontinue Motrin and the patient should have an ophthalmologic examination, including central visual fields.

Fluid retention and edema have been associated with Motrin; use with caution in patients with a history of cardiac decompensation

Motrin can inhibit platelet aggregation and prolong bleeding time. Use with caution in persons with intrinsic coagulation defects and those on anticoagulant therapy.

Patients should report signs or symptoms of gastrointestinal ulceration or bleeding, blurred vision or other eye symptoms, skin rash, weight gain, or edema.

To avoid exacerbation of disease or adrenal insufficiency, patients on prolonged corticosteroid therapy should have therapy tapered slowly when Motrin is added.

Drug interactions. *Aspirin* used concomitantly may decrease Motrin blood levels. *Coumarin:* Bleeding has been reported in patients taking Motrin and coumarin.

Pregnancy and nursing mothers: Motrin should not be taken during pregnancy or by nursing mothers.

Adverse Reactions

Incidence greater than 1%

Gastrointestinal: The most frequent type of adverse reaction occurring with Motrin is gastrointestinal (4% to 16%). This includes nausea,* epigastric pain,* heartburn,* diarrhea, abdominal distress, nausea and vomiting, indigestion, constipation, abdominal cramps or pain, fullness of the GI tract (bloating and flatulence). **Central Nervous System:** Dizziness,* headache, nervousness. **Dermatologic:** Rash* (including maculopapular type), pruritus. **Special Senses:** Tinnitus. **Metabolic:** Decreased appetite, edema, fluid retention. Fluid retention generally responds promptly to drug discontinuation (see PRECAUTIONS).

*Incidence 3% to 9%.

Incidence less than 1 in 100

Gastrointestinal: Upper GI ulcer with bleeding and/or perforation, hemorrhage, melena. **Central Nervous System:** Depression, insomnia. **Dermatologic:** Vesiculobullous eruptions, urticaria, erythema multiforme. **Cardiovascular:** Congestive heart failure in patients with marginal cardiac function, elevated blood pressure. **Special Senses:** Amblyopia (see PRECAUTIONS). **Hematologic:** Leukopenia, decreased hemoglobin and hematocrit.

Causal relationship unknown

Gastrointestinal: Hepatitis, jaundice, abnormal liver function. **Central Nervous System:** Paresthesias, hallucinations, dream abnormalities. **Dermatologic:** Alopecia, Stevens-Johnson syndrome. **Special Senses:** Conjunctivitis, diplopia, optic neuritis. **Hematologic:** Hemolytic anemia, thrombocytopenia, granulocytopenia, bleeding episodes. **Allergic:** Fever, serum sickness, lupus erythematosus syndrome. **Endocrine:** Gynecomastia, hypoglycemia. **Cardiovascular:** Arrhythmias. **Renal:** Decreased creatinine clearance, polyuria, azotemia.

Overdosage: In cases of acute overdosage, the stomach should be emptied. The drug is acidic and excreted in the urine, so alkaline diuresis may be beneficial.

Dosage and Administration: Rheumatoid and osteoarthritis, including flares of chronic disease: Suggested dosage is 300, 400 or 600 mg t.i.d. or q.i.d.

Mild to moderate pain: 400 mg every 4 to 6 hours as necessary for relief of pain.

Do not exceed 2400 mg per day.

Caution: Federal law prohibits dispensing without prescription.

For additional product information, see your Upjohn representative or consult the package insert.

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August, 1979

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VITA-STAT PROFILE: HYPERTENSION

by Ralph E. Snyder, M.D., F.A.C.P. / Medical Director

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Dr. David M. Berkson Chicago Department of Health re. study on Vita-Stat Computer
May 1979 American Journal of Public Health

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Joseph H. Chadwick, Ph.D., Director, Health Systems Programs
SRI International

"Blood pressure measurements by the Vita-Stat correlated as well as did measurements by physicians and provided reliable determinations suitable for the purposes of screening, referral and monitoring of treatment."

Dr. R. Patterson Russell, Director of the Hypertension Clinic at Johns Hopkins Hospital and Chairman of the High Blood Pressure Commission of Maryland
March, 1979 "Preventive Medicine"

***Copies of these and other reports, or medical information about the Vita-Stat Computer is available by writing to me:
Ralph E. Snyder, M.D., Medical Director
2801 75th Street North, St. Petersburg, Florida 33710.

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Bernard E. Levine, M.D.
Richard M. Riedy, M.D.

Editors:
Faysal M. Hasan, M.D.
Richard I. Lemen, M.D.
Bernard E. Levine, M.D.

Sleep has always been regarded by scientists and laymen alike as a time of renewal, of recovery from physical and mental ills. However, if sleep itself is pathologic, patients may manifest clinical problems during waking hours which correlate directly with the degree of sleep disruption. The nature of the waking abnormality cannot be understood until its relationship to sleep pathology is clarified. The interrelation of the Pickwickian Syndrome and sleep apnea is an interesting case in point. In 1912, Sir William Osler noted, "a remarkable phenomenon associated with excessive fat is an uncontrollable tendency to sleep, like the fat boy in Pickwick". In 1956, Burwell picked up on the reference to Dickens and coined the term "Pickwickian Syndrome" to describe the combination of obesity, hypersomnolence, alveolar hypoventilation, and cor pulmonale. Over the next number of years many studies were performed without discovering a pathophysiologic explanation for this symptom complex. Then in 1966, Gastaut noted that these patients would recurrently stop breathing during sleeping hours. By 1972, at a symposium in Italy, a clear pattern of sleep apnea syndrome was delineated. Since that time, data has rapidly accumulated demonstrating not only that sleep apnea is the major disorder underlying the Pickwickian Syndrome, but that the problem of sleep apnea is a much broader one and that it represents a common, significant clinical entity.

Everyone has brief periods of apnea during sleep. Sleep apnea syndrome is defined as the occurrence of 30 or more episodes of apnea lasting greater than 10 seconds during 7 hours of nocturnal sleep. Symptomatic patients with sleep apnea syndrome may stop breathing hundreds of times during a single night with episodes of apnea lasting between 20 and 90 seconds. On an average, these patients are apneic during 50% of sleep time.

Sleep apnea may be classified as obstructive, central, or mixed. Obstructive apnea is the most common occurring in 85-95% of various series. Obstructive apnea is characterized by persistence of diaphragmatic and chest wall movement but lack of effective airflow at the nose and mouth because of upper airway obstruction. In children, this is usually because of hypertrophied tonsils or adenoids. In adults, it is caused by the closure of the posterior pharynx due to intermittent loss of tone of the tongue and pharyngeal musculature. The cause for this muscle relaxation is unknown. Symptomatic obstructive apnea seems to be increased by age and obesity. Anatomic abnormalities such as micrognathia also predispose to obstructive apnea.

Central apnea occurs secondary to arrest of respiratory automaticity. There is no chest wall motion and no airflow. Central apnea may be associated with a history of poliomyelitis, brain stem disease, or spinal surgery. Usually no know predisposing factor exists. Sudden infant death syndrome is felt by many to represent a form of central sleep apnea.

A significant number of patients demonstrate combined obstructive and central or mixed apnea. The inter-relationship between central and obstructive problems suggest that both may be manifestations of abnormal respiratory control or abnormal arousal mechanisms.

The presenting complaint of sleep apnea patients may very markedly. Patients referred to specialized sleep centers usually have predominant complaints of excessive daytime sleepiness, whereas patients seen

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From: Good Samaritan Hospital, Pulmonary Function Laboratory, 1033 E. McDowell Road, Phoenix, AZ 85006. (Dr. Levine, Director, Pulmonary Function Laboratory.) (Dr. Riedy, Director, Sleep Evaluation Section.) Reprint requests to Dr. Levine.

initially in cardiopulmonary practice present unexplained respiratory failure, cor pulmonale, or cardiac arrhythmia. In the latter group, patients and physicians may not connect waking symptomatology to nighttime sleep patterns.

Symptoms of sleep apnea are caused by airflow obstruction, sleep deprivation, and apnea-related hypoxemia. Symptoms of obstruction are often dramatic. Loud snoring is a universal complaint. Snoring has usually been a problem for years and may be progressing at the time of diagnosis. Snoring may well make sleep impossible for the patient's spouse. During periods of complete obstruction there may be a brief pause in the snoring sound and then the patient will resume breathing with a loud snort. Arousals may also be attended with gasping and choking. Abnormal motor movements, including major movements of arms and legs may also occur during apnea and may cause great distress, possibly physical harm to the bed partner. Because of loud snoring and motor movements a common history is that the patient and his wife sleep in separate beds or separate rooms. In contrast to the abnormal motor activity, another cause of anxiety for the spouse may be intermittent cessation of breathing during sleep.

Nighttime symptoms also may include enuresis. Impotence has been noted in large number of patients.

Patients with sleep apnea have marked disruption of sleep patterns with decrease in Stage III, Stage IV, and REM sleep. There is a marked increase in arousals during the night and in the amount of time spent in an awake or near awake state. Not surprisingly then, a major daytime symptom of sleep apnea is excessive sleepiness. This may be simple fatigue or may be sleepiness so severe that the patient is unable to drive a car or carry on a conversation without falling asleep. Patients may demonstrate a decrease in intellectual capacity. Indeed, in severe cases, patients may demonstrate automatic behavior, being able to carry out simple repetitive tasks but being unable to maintain complex behavior patterns. Personality changes may be noted. Irritability, hostility, and paranoia may be present.

Recurrent nighttime apnea leads to long periods of severe hypoxemia and arterial hemoglobin desaturation. Hypoxemia is a potent stimulus to increased pulmonary arterial pressure. Right ventricular hypertrophy, cor pulmonale, and congestive heart failure may develop over time. Long periods of exposure to low levels of oxygen tension will also blunt hypoxic drives and may lead to daytime hypoventilation.

During periods of obstruction there will be marked swings in intrathoracic pressure as the patient attempts to breathe. These variations will lead to changes in pulmonary arterial pressure which may increase right heart symptomatology. Beyond that, they will lead to increased parasympathetic discharge with secondary slowing of cardiac rate. A characteristic feature of sleep apnea may be marked sinus arrest for periods up to 12 seconds in duration. Coincident with termination of apnea, there will be a sympathetic release associated frequently with ventricular arrhythmias varying from isolated premature ventricular contractions to runs of ventricular tachycardia. It is clear form a consideration of the cardiac responses to sleep apnea that these patients are at high risk for sudden death.

Some of the physical features of sleep apnea patients have already been reviewed. For reasons that are not entirely clear, most obstructive apnea patients are males. This predominence may relate to the anatomic configuration of the male pharynx or there may be actual differences in respiratory control between sexes.

Transient increases in systemic blood pressure occur during apneic episodes. Over time, blood pressure elevation becomes sustained and patients may present with significant arterial hypertension. As noted before, pulmonary artery pressure may also be increased and patients may present with classic cor pulmonale.

While many of the patients are obese, and some massively, obesity is not always present and its exact relationship to the development of apnea is not fully understood. Historically, patients report variable symptomatology with worsening and improvement of symptoms over many years. Weight gain, aging, and decrease in general health often lead to a worsening of symptoms.

Once sleep apnea is suspected patients may be assessed by simultaneous monitoring of a number of physiologic parameters during sleep. These include nasal airflow, chest wall movement, arterial saturation by ear oximetry, electroencephalogram, electro-occulogram, electromyogram, and electrocardiogram.

Screening techniques of sleep apnea appropriate for use in the community hospital have been reported using either simple methods of measuring nasal airflow and chest wall movement or the use of a Holter monitor to demonstrate marked sleeping sinus arrhythmia.

Treatment depends upon the severity of symptomatology and the pathophysiological consequences of hypoxemia. Weight loss must be stressed and attempted, but is often difficult to achieve. In mild cases, medications may be tried. Progesterone has been effective in some cases. In more severe cases, when life threatening arrhythmias or marked depression in hypoxic and hypercapneic drives import a much more serious prognosis, tracheostomy may be necessary. The tracheostomy is closed during daytime hours to allow normal function and speech, and opened during sleep to bypass the obstruction.

In the central type apnea, medications have also been tried. The most effective to date has been progesterone. This drug works as a non-specific ventilatory stimulant. Tricyclics, particularly Vivactil, have been tried with only transient effect. Aminophylline, although effective in infants with central type of apnea, has had little effect in adults with sleep apnea syndrome. In central apnea, ow-flow oxygen has also been used with success. In evere cases, electrophrenic stimulation may be xtremely rewarding. This treamtent invloves mplantation of a small platinum electrode around the hrenic nerve. This electrode is attached to a radio eceiver implanted into the chest wall usually in the ectoral region. During the night, an antenna is placed ver the radio receiver and attached to a power source vhich delivers a radio frequency signal at a preset rate nd duration of stimulus.

No data exists at this time on a true incidence of sleep pnea. Data indicate that 31% of males and 19% of emales demonstrate habitual snorning; 6% of these abitual snorers will have significant sleep related espiratory complaints. Studies also show that many symptomatic individuals exist who have significant pnea and arterial desaturation during sleep. Apparently susceptible individuals tolerate sleep apnea for many years until aging, weight gain, or illness causes clincial symptomatology. It is of interest that a number of instances of familial sleep apnea have now been reported.

Recent studies have shown apnea or hypopnea with marked oxygen desaturation may occur during sleep in patients with chronic obstructive lung disease. Indeed, patients with satisfactory daytime oxygen levels who have unexplained cor pulmonale or polycythemia must be suspected of significant hypoxemia during sleep. Sleep monitoring studies may be very important in this patient group.

In summary, sleep apnea syndrome is a recently described entity with possible consequences of severe psychological disability, right heart decompensation, and sudden death. A high level of clinical suspension and a careful sleep history can quickly lead to appropriate diagnosis and therapy.

Arizona Lung Association Self-Teaching Materials

Robert W. Shook

All Arizona physicians are invited to utilize the self-teaching materials of the Arizona Lung Association and its medical section, the Arizona Thoracic Society.

The Arizona Lung Association has announed that it has acquired eight new audio-visual programs and is making them available on loan to physicians and other health professionals. Prepared for the American Thoracic Society Learning Resources Center, and reviewed by the ATS Component Committee on Audio Visual, these are slide-tape shows, each about 25-35 minutes in length. Included in each program are slides, audio-cassette tape, and typescript of the text.

These audio-visual programs are in addition to the many papers, articles, and brochures on lung diseases maintained by the Arizona Lung Association. Some are designed for the general public and are ideal for the literature rack in physicians' offices. Others are specifically for the physician.

For further information on available literature, or to borrow the new audio-visual programs, call either the Phoenix office at 258-7505, or the Tucson office at 323-1812.

The eight audio-visual programs currently available are:

Basic Pulmonary Function Testing

This program reviews lung function specifically as it pertains to the measurement of the functional capability of the lungs; describes which tests are most useful in clinical practice (including spirometric tests but not including the measurement of lung volumes or tests of lung mechanics); lists the most common indications for doing pulmonary function tests; and describes briefly the method of testing and the interpretation of results. It is excellent for introducing the concepts of pulmonary function testing.

The Pathophysiology of Pediatric Asthma

This program describes the pathophysiology of pediatric asthma with emphasis on the between-attack period and the role of small airways in the process. At the conclusion of this program, the viewer should be able to state the four pathophysiologic elements of asthma; identify the site of increased airway resistance; and describe the causes of air trapping and the increased work of breathing.

From: Arizona Lung Association, 1239 E. McDowell Road, Phoenix, AZ 85006. (Mr. Shook, Program Director)

Interpretation of Blood Gases

This program approaches interpretation of clinical blood gas measurements from a physiologic viewpoint, emphasizing abnormal mechanisms that produce hypoxemia and hypercapnia. Understanding of the underlying pathophysiology is stressed. Patient examples are used to illustrate the importance of blood gases in diagnosis and treatment. Physiologic principles of oxygen therapy are included in the discussion of case material.

The Measurement of Closing Volume

The earliest changes of chronic airways obstruction take place in small airways. Detection of these manifestations of disease has received increasing attention recently. The closing volume (the lung volume at which dependent lung zones cease to ventilate) is one of several newer tests currently under study as a diagnostic tool. In this balanced presentation, the author summarizes the theory on which the test is based; describes the standardized procedure for measurement; and discusses possible uses and limitations of the test.

Mycobacteria Other than M. Tuberculosis in Man

Through excellent narration and photography, the author reviews: The taxonomy of mycobacteria other than *M. tuberculosis*; the clinical and roentgenographic characteristics of the mycobacteria; the cultural characteristics that differentiate them; and the epidemiology of such infections. He suggests an approach to treatment based on extensive experience. Of particular value is a summary using a series of questions and answers to reinforce the major points made in the program.

Pathophysiology of Pulmonary Edema

The recognition, pathophysiology and principles underlying treatment of pulmonary edema is presented. The program discusses fluid balance in the lung under normal and abnormal circumstances, and divides pulmonary edema into two broad categories: high pressure edema and increased permeability edema, depending on the underlying mechanism. Specific examples and principles of managment of both forms of edema are presented.

Pathology of Chronic Airflow Obstruction

The pathology of chronic bronchitis and emphysema is discussed, pointing out the contrasting features of these two entities. Emphysema is subcategorized on a morphologic basis. Slides include both gross and microscopic lung sections.

Pediatric Segmental Bronchial Drainage

The segmental anatomy of the lung is described and correlated with surface and roentgenographic anatomy. Segmental bronchial drainage techniques in children are demonstrated and related to bronchi anatomy. The indications, precautions and contraindications are presented. The specific problems of drainage in children are emphasized.

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Seminars in
Gastroenterology
and Liver Disease

Antibiotic-Associated Pseudomembranous Colitis

Rapid Clinical Response to Oral Vancomycin

George N. Kerrihard, M.D.

Editors:
George Burdick, M.D.
Stephen Glouberman, M.D.
Leon Rigberg, M.D.

Abstract

Three cases of antibiotic-associated pseudomembranous colitis are reported with rapid clinical improvement following oral vancomycin. Toxin-producing Clostridium difficile was isolated from stool specimens in two patients both of whom also showed rapid clearing of the pseudomembranes.

Introduction

Antibiotic-associated pseudomembranous colitis is a well recognized entity whose clinical presentation varies from mild diarrhea to fulminate colitis. Most cases respond to supportive care and discontinuation of suspected antibiotic. Some, however, continue to have persistent disabling diarrhea, toxemia, protein-losing enteropathy, ileus and in extreme cases, colonic perforation or death.[1, 2]

Steroids or cholestyramine have been utilized with some success.[3, 4, 5] Subtotal colectomy has been necessary in severely ill patients.[6]

The following cases of antibiotic-associated pseudomembranous colitis are reported showing rapid clinical improvement with oral vancomycin.

From: Tucson Medical Park, 5302 E. Grant Road, Tucson, AZ 85712

Case I

A 75 year old woman was hospitalized at St. Joseph's Hospital on 1/16/78 after 14 days of persistent diarrhea. She noted the onset of 6 to 8 watery bowel movements a day following two weeks of ampicillin therapy for a pulmonary infection. Sigmoidoscopy with rectal biopsy showed classical pseudomembranous colitis. In spite of I.V. fluids and ten days of Prednisone therapy up to 80 mg. per day, her diarrhea increased to 12 stools a day. After 24 days of diarrhea, steroids were stopped and oral vancomycin, 500 mg. Q 6 h., was instituted based on reports of its effectiveness in hamsters with clindamycin induced colitis. Her diarrhea subsided dramatically. Within 24 hours she was having one bowel movement a day. Vancomycin was continued for five days and patient was discharged free of symptoms.

Case II

A 62 year old woman was admitted to Tucson Medical Center on 2/1/78 for excision of a pulmonary lesion. One month prior she developed a cough and fever. A chest x-ray showed a left lower infiltrate which failed to clear after two weeks of phenoxymethyl penicillin, 400 mg. Q.I.D. One week prior to admission she noted the onset of watery diarrhea. A lobectomy performed on 2/2/78 showed a pseudolymphoma. Postoperatively, she developed daily temperature spikes to 102 and 103 in spite of I.V. Keflin, 500 mg. Q 6 h. After eight days of fever and 15 days of 6-8 watery bowel movements per day, she developed marked abdominal distention and tenderness. White count rose to 21,500 with 8 bands, 80 segs. Total proteins decreased to 4 gm. with albumin 2.1 gm. Pseudomembranous plaques were felt on rectal examination. Sigmoidoscopy with biopsy confirmed severe extensive pseudomembranous colitis. N.G. suction was started along with N.G. tube instillation of 500 mg. vancomycin Q 6 h., with the N.G. tube clamped for one hour after each dose. Vancomycin was also given I.V., 500 mg. Q 6 h. Within 24 hours her temperature was normal and her diarrhea subsided. Sigmoidoscopy three days later showed virtual clearing of her entire rectosigmoid with only two small plaques remaining. Patient received I.V. vancomycin for three days and oral vancomycin for five days. She was discharged having one normal bowel movement a day. Stools obtained prior to vancomycin therapy were sent to Dr. Vera Sutter's laboratory at Wadsworth VA Hospital where toxin-producing Clostridium difficile was isolated.

Case III

A 78 year old man was admitted to Tucson Medical Center on 3/22/79 for massive gastrointestinal bleeding secondary to aortoduodenal fistula. Following surgery requiring 20 units of blood, he was placed on I.V. Kefzol, 500 mg. Q 4 h., for four days and then switched to Cefadyl, 2 gm., I.V. Q 4 h. On the 10th postoperative day he began having severe diarrhea. Twelve watery stools a day were noted along with fecal incontinence and weakness. Total proteins decreased to 4.4 gm. with albumin 1.9 gm. Palpable plaques were noted on rectal examination. Typical pseudomembranous colitis was confirmed on sigmoidoscopy and rectal biopsy. Cefadyl was discontinued and oral vancomycin, 500 mg. Q 6 h., was started with prompt cessation of diarrhea. Sigmoidoscopy five days later was normal. Vancomycin was continued for six days. Stool specimens sent to Dr. Sutter's laboratory showed the presence of both Clostridium difficile and its toxin.

Results

Three cases of antibiotic-associated pseudomembranous colitis are reported. A rapid clincial response was noted in all following oral vancomycin. In two cases toxin-producing Clostridium difficile was isolated and rapid clearing of the pseudomembranes was documented. All patients tolerated oral vancomycin well and none relapsed.

Discussion

Antibiotic-associated pseudomembranous colitis is a well described clinical pathologic entity with characteristic morphologic and histologic findings.[1,7]

Early case reports were noted following Aureomycin and chloramphenical.[7] In 1970 an increased incidence was reported with lincomycin therapy.[8] Later a flurry of articles showed a high association in conjunction with clindaycin.[9,10] Presently a wide variety of antibiotics have been implicated.[2,11]

Its etiology remained an enigma until Larson[11,12] discovered a cytotoxin in the stools of patients with antibiotic-induced pseudomembranous colitis. A similar fecal toxin was isolated by Rifkin.[13] Rifkin[14] and Bartlett[15] also isolated a toxin in hamsters with clindamycin-induced colitis. All studies suggested a clostridial organism. Subsequent studies by Bartlett[16] and George[17] isolated Clostridium difficile as the toxin-producing organism.

Bartlett[18] demonstrated the protective effect of vancomycin in hamsters. An early clinical report suggested the efficacy of oral vancomycin.[13] Tedesco[19] reported rapid clinical responses in nine patients receiving oral vancomycin. All showed decrease in stool toxin levels and proctoscopic clearing. In one patient treated with I.V. vancomycin the clinical response was difficult to evaluate. No decrease in toxin titers were observed and vancomycin was undetected in the stool, suggesting the importance of oral administration.

Antibiotic therapy apparently alters the colonic flora allowing the emergence of toxin-producing C difficile.[20] This toxin produces the characteristic clinical and pathologic findings in this disease. Results with oral vancomycin indicate it to be an effective form of therapy.

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Infectious Disease

An Occurrance of Q Fever in Yuma County

A Sporadic Case of *Coxiella Burnetti*

H. Peter Hernried, M.D.
Douglas K. Perkins, M.D.

Editors:
Peter Kelly, M.D.
Peter P. McKeller, M.D.
Phillip J. Rubin, M.D.

Abstract

An endogenous case of Q fever in Arizona is reported, the first such case known. The diagnosis was established in the face of a rising titer of the *Proteus* OX19 antigen, the Felix-Weil reaction, which is characteristically negative in Q fever, unlike in other rickettsial diseases. It is emphasized that this entity must now be considered in differential diagnoses in Arizona.

Introduction

Since the first description of Q fever in Australia in 1937, the disease has obtained a worldwide distribution.[1] Q fever has twice previously been diagnosed in Arizona, but was thought in both cases to have been contracted elsewhere.[2]

This paper documents a confirmed case of endogenous *Coxiella Burnetti* infection contracted in Yuma County in an agricultural setting. It will further describe the unexpected diagnosis of this entity during the workup of a fever of unknown etiology, based on positive Felix-Weil reaction, a *B. Proteus* OX19 antigen, this test being typically negative in Q fever.[3] Negative rickettsial titers were subsequently obtained in the patient's family and sheep.

From: St. Joseph's Hospital and Medical Center, 350 W. Thomas Road, P.O. Box 2071, Phoenix, AZ 85001.

History of Q Fever

"Query" fever was first described by E. H. Derrick in 1937, when he reported nine cases diagnosed on the basis of guinea pig inoculation and by the demonstration of immunity.[4] These cases occurred in meat industry workers in Brisbane, Australia where an unusual febrile illness had been recognized since early in 1933. The disease was described as a clinical entity of acute onset, consisting of headache, malaise, anorexia, myalgias, fever, relative bradycardia, rigors, and the the notable absence of rash, jaundice, or splenomegaly. Derrick, unable to identify a causative organism, sent guinea pig livers to Dr. F. M. Burnet, of Melbourne, who transferred the infection to mice and discovered rickettsial bodies in their spleens.

H. Noguchi had, in 1926, reported recovering a filter-passing "virus" from a North American tick, *Dermacentor Andersoni*, and succeeded in infecting guinea pigs and monkeys.[5] Twelve years later and some sixty miles away, Gordon E. Davis and Harold R. Cox isolated a similar filter-passing infectious agent from *D. Andersoni*, near Missoula, Montana, and were unable to demonstrate by cross immunity tests any relationship to Rocky Mountain spotted fever, epidemic typhus, or any other rickettsial disease known to be in North America.[5]

Dyer described a case of human infection by this organism in a staff member of the National Institute of Health, who had spent a few days in a Public Health Service laboratory in Hamilton, Montana, where the infection was being studied in animals.[6] The staff member developed a febrile illness, manifesting symptoms of retro-orbital headaches, malaise, chills, arthralgias, and a relative bradycardia. It is interesting that this individual had a positive *B. Proteus* OX19 antigen at the low titer of 1:80 complete, and 1:60 partial, though his serum had previously been positive, and he had had typhus fever six years prior.

Q fever is now recognized as an acute self-limited illness caused by the inhalation of rickettsial bodies. Recognized complications of Q fever include uveitis, optic neuritis, meningitis, and congenital malformations. Chronic Q fever has been associated with such complications as pleuropericarditis, thrombophlebitis, and endocarditis (which is considered uniformaly fatal). A common complication is hepatocellular dysfunction which appears histologically as an extensive granulomatous hepatic infiltration. Histologic changes in Q fever hepatitis consists of nonspecific granulomatous hepatitis distinctly different from a viral hepatitis both in the histopathology of the inflammatory lesion and in the fatty infiltration of the noninflamed hepatocytes. The histopathology is usually more impressive than the associated elevations in liver function tests. Though respiratory symptoms are common, with some investigators reporting pneumonitis in as many as fifty percent of their cases,[8] severe pneumonia is rare.[9]

Epidemiology of Q Fever

The most important reservoirs of *Coxiella Burnetti* ar ticks, farm animals, and wildlife. Livestock, whil carriers of the rickettsia, have not been shown t experience any ill health as a result of infection. Th human route of infection is most commonly vi inhalation or ingestion. Tick bites may be responsible fo other mammalian infections, however, they have neve been shown to cause infections in humans, though inhalation of tick feces might produce such infection.[1]

The outbreak in Brisbane has already been described as has the single early North American occurrence in laboratory worker. The first reported cases of naturally occurring Q fever in the northern hemisphere occurre among troops in Italy during the Second World War Antibodies were found in the sera of the loca population. Q fever was reported in Texas in 1947 and i California in 1948. During the 1950s reports sprea northward in the United States, with cases occurring in Maryland and British Columbia in 1959. By 1953 cases had been reported over a wide area of Great Britain.[1]

The incidence and geographical distribution of Q fever in the United States at this time is unknown. Data are incomplete since Q fever is an optionally reported disease.[10] Prior to the case herein reported, the Arizona Department of Health is aware of only two serologic specimens obtained by Arizona physicians which showed a significant rise in titer of the complement fixing Q fever antigen. In one case the disease was thought to have been imported from Australia, and in the other, the entity was felt to have been contracted in New Mexico.[2]

Case Presentation

Subjective: A 43-year-old construction worker, a life-long resident of Yuma County, presented with a fever and a headache of one week's duration.

The patient was in excellent health until 7/20/77, on which evening he vomited and began to experience a severe frontal headache. During the night, the patient developed shaking chills, which lasted about two hours. The next day the patient went to work in spite of recurrent chills and persistent cephalalgia. The following day, the patient remained in bed. The patient experienced no more nausea or vomiting, but he developed complete anorexia. He was seen by a physician on 7/24/77, who gave an intramuscular injection of penicillin and some codeine. The following morning, the patient was admitted to Yuma Regional Medical Center with a temperature of 102°F., and a regular pulse of 70. His blood pressure was 102/60 millimeters of mercury. The patient's physical examination at that time was otherwise unremarkable.

Throughout the patient's hospital stay in Yuma, his fever persisted and was lowered only with the use of antipyretics. He continued to suffer from headache. He denied change in stool color or dysuria. The patient had no arthralgia and no upper or lower gastrointestinal symptomatology. The patient suffered no pulmonary complaints and the remainder of the review of systems was negative, except that he did notice a darkening of urine.

The patient's past medical history was unremarkable, there being no history of tuberculosis, coccidioidomycosis, skin tests thereof, or rheumatic fever. The patient was a construction worker who lived on a farm near Somerton, Arizona. He denied any physical contact with livestock, though his son raised sheep on the farm. Neither did the patient handle agricultural chemicals, though he did not exclude the possibility that he may have consumed contaminated water. The patient had not traveled to nearby Mexico, nor to any other foreign or tropical countries. He had never been immunized against typhus. The patient was a nonsmoker and a social drinker.

Objective: Physical Examination - On admission to St. Joseph's, the patient had a fever of 101.2°F., a regular pulse of 84, a blood pressure of 130/90 millimeters of mercury in the left arm, and 16 effortless respirations per minute. In general, the patient was a slightly overweight, muscular, well-developed male, who was alert, oriented, and cooperative. He did not appear in any acute distress at the time of the initial examination. His skin was warm, dry, and normally hydrated, with no sign of rash or other skin lesion. Sclerae and conjunctivae were clear, and there was no icterus. The neck was supple, and examination was otherwise negative to inspection, ausculatation, and palpation. No generalized or regional lymphadenopathy was present. The thorax was normal to palpation inspection, percussion, and auscultation. The precordium was inactive and heart tones were entirely normal. The abdomen was soft and there was no tenderness, visceromegaly, or abnormal mass. Bowel sounds were normal. No costovertebral angle or perispinal tenderness could be appreciated. Examination of the genitalia and rectum were normal. Muscle tone and mass were appropriate, and there was no muscular tenderness.

Laboratory Data - at the time of the patient's presentation at St. Joseph's certain laboratory data were available from Yuma, including a normal spinal fluid analysis, urinalysis, and monospot test. Electrolytes were normal, and a CBC showed a normal hemoglobin and a white count of 5,200, with 36 neutrophils, 19 bands, 1 eosinophil, 17 lymphocytes, and 27 monocytes, with adequate platelets. The patient had a normal alkaline phosphatase, but the SGOT was elevated to 50 U/L, and the SGPT at 120 U/L. Negative cultures were obtained from the urine, CSF, and throat swab. A brain scan, EEG, and skull series were all within normal limits. A chest x-ray was normal.

Hospital Course - The patient was transferred via ambulance to St. Joseph's Hospital's Neurosurgical Service on 7/27/77 with the chief complaint of severe headache and fever. The headache resolved soon after admission to St. Joseph's but a chronic intermittent fever and anorexia persisted. The patient received forty eight hours of I.V. Ampicillin at a dose of 2 grams every four hours for a possible salmonella infection. There was no response, and the antibiotic was discontinued. Blood and urine cultures later proved to be negative. The patient's sedimentation rate was elevated at 35 millimeters per hour, and his total white count remained normal. ANA and RA were negative, and the C_3 was normal. No hepatitis associated antigen was found. Spherulin and PPD skin tests were negative, with a positive control. No ova or parasites were discovered upon repeated stool examinations and leukocytes were not found. A fixed smear for malaria disclosed no parasites. A coagulation profile and a serum protein electrophoresis were unremarkable. Febrile agglutinins showed only a 1:40 titer to the *Proteus* OX19 antigen. Repeat febrile agglutinins were again negative for typhoid and Brucella antigens, but a rise in titer to 1:320 was documented for the *Proteus* OX19 antigen. Serum was sent for rickettsial titers to the Center for Disease Control.

Hepatic enzymes remained persistently elevated, including alkaline phosphatase and total bilirubin. Oral cholecystogram, intravenous pyelogram, upper G.I. series, and a sigmoidoscopy all were within normal limits. A liver-spleen scan showed diffuse hepatocellular dysfunction. At this point a liver biopsy was obtained which showed granulomatous hepatitis with mild fatty changes.

Ultimately, results were returned from the CDC showing a diagnostic rise in the Q fever antigen from 1:128 acute, to 1:1024 convalescent. The patient was begun on a course of oral chloramphenicol after being appropriately warned of the risk of bone marrow suppression. The fever resolved promptly. The patient was followed for three afebrile days while therapy was continued. During this time, symptoms resolved completely, though some residual weakness and weight loss were evident. The patient was discharged on 8/16/77 on oral tetracycline.

The titers for Rocky Mountain spotted fever rose from 1:8 to 1:128 and for murine typhus from 1:16 to 1:128. These changes were not considered diagnostic in light of the much higher rise in titer of the Q fever antigen.

Assessment

The greater than fourfold rise in titer of specific Q fever antigen, along with the prompt response to treatment, suggested an acute *Coxiella Burnetti* infection.[8] The rise in *Proteus* OX19 titer is interesting in that Q fever is the one rickettsial group which is not cross reactive with any *Proteus* antigen.[3] False positive responses, however, are seen in severe liver disease, leptospirosis, *Borrelia* infections, and *Proteus* urinary tract infections.[11] We have no definite explanation for the rise of OX19 antibody, though it may have been related to liver disease. Nor is there an explanation for the slight rise in titer of Rocky Mountain spotted fever and murine typhus complement fixation, though the rise was not greater than fourfold in either case. Following the patient's return to Yuma County, sera were drawn from the family members and from the sheep which grazed on his land. No significant titers were found in either human or bovine specimens.

Conclusions

This paper has documented a sporadic occurrence of Q fever in Arizona. Thus, Q fever must now be considered in the differential diagnosis of fevers of unexplained etiology in that state. Other presentations must also be expected, such as headache, jaundice, and atypical pneumonia. The case herein presented also documents that a positive Felix-Weil reaction need not exclude consideration of Q fever among other rickettsial infections. In this case, no human or bovine reservoir of rickettsia could be demonstrated on the basis of serologic titers. It is emphasized that the diagnosis of Q fever is especially important in light of the good response of the disease to treatment.

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Obstetrics and Gynecology

A Practical Guide to Sexual Counseling

Kathryn L. Reed, M.D.
Louis Weinstein, M.D.

Editors:
Walter B. Cherny, M.D.
Louis Weinstein, M.D.

"Sex is a natural need, like the need for sleep, warmth, air, food. . .yet it is, in addition, the only body process which can be voluntarily and permanently controlled without threat to the life of the individual, as would prevention of breathing, elimination, or eating."[1]

Different forms of sexual therapy have probably been in existence as long as the human has been motivated to improve his plight. During the past quarter century, a renewed interest in sexual counseling has occurred and today it is available on an ever-increasing scale.

Prior to this current social revolution, sexuality was approached as something which was instinctive and therefore, to be left out of the realm of words, teaching or analysis. Fortunately, the Victorian view of sexuality is disappearing and scientific investigations of sexual behavior are currently being performed.

In 1967, figures were compiled by Donald Burnap and Joshua Golden[2] regarding the frequency with which the average practicing physician encounters patients with sexual problems. In the review, the types of sexual problems in a decreasing order of frequency were: lack of orgasm, frigidity, frequency of intercourse, general sex information, impotence, dyspareunia, lack of affection, premarital counseling, extramarital

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From: Department of Obstetrics and Gynecology, University of Arizona Health Sciences Center, Tucson, AZ 85724.

intercourse, and premature ejaculation.

Frank et al[3] reported that a minimum of 50% of all married couples are troubled by sexual problems. In this study, 40% of the men reported erectile or ejaculatory dysfunction and 63% of the women reported arousal or orgasmic dysfunction.

One definition of sexual dysfunction is impaired, incomplete or absent expressions of normally recurrent human sexual desires and responses. Masters and Johnson,[4] in *Human Sexual Inadequacy*, list the major forms of sexual dysfunction:

Female, anorgasmia (inability to achieve orgasm)
  1. primary - lifelong
  2. secondary - formerly orgasmic
     hyporgasmia or infrequent orgasm
     dyspareunia - painful coitus
     vaginismus
Male, erectile failure
  1. primary
  2. secondary
     premataure ejaculation
     ejaculatory incompetence

Physical ailments will be present in 5-10% of patients with sexual dysfunction while a similar percentage will have deepseated psychological problems requiring psychiatric evaluation. The average practitioner can offer valuable assistance to the patient with a sexual problem. The physician must be open to the needs of the patient and not allow personal views to interfere with patient care. A sexual history is obtained during the initial interview, along with a description of any sexual problems. Details to be included are onset and course of the dysfunction, what improves or worsens the problem, the patient's assessment of the problem, methods of resolution attempted, and the goal of treatment. Openness can be communicated in a variety of ways from office discussions to questionnaires filled out in private. Some physicians have had success with compiling their own manual of normal sexual function and the basic skills important for sexual health. Treatment consists of providing information related to sexual behavior, performing a thorough physical examination, and assisting with development of communication skills.

There is a tremendous need for the physician to provide sexual counseling for the average patient. This requires a skill which can only be developed by familiarity with the subject and communication with the patient.

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Oncology

The Role of Radical Neck Dissection in Head and Neck Carcinoma

Charles F. Koopman, Jr., M.D., M.S.
Stanley W. Coulthard, M.D.

Editor:
David Alberts, M.D.

Introduction

In the development of a treatment plan for the patient with a head and neck malignancy, the head and neck surgeon must carefully evaluate: the probability of regional nodal metastasis, the effect of surgery on the patient's way of life (occupation), the results of the alternatives such as radiation or chemotherapy, and the complications of surgery.

When the cervical metastases are initially readily palpable, or when adenopathy develops after primary resection or irradiation, there is usually concurrence concerning the efficacy of a radical neck dissection (hereafter, RND). However, all too often, the mention of a RND is met with fears of a severely deforming or crippling procedure poorly tolerated by the patient. Also, the topic of an elective neck dissection stirs much controversy, especially in this era of aggressive radiation oncology services. Thus, in this presentation, we shall attempt to discuss our philosophies about technique, to mention modifications to the standard RND, to evaluate data on the value of elective, therapeutic, and salvage RND's, and to discuss the complications of the procedure.

From: Sect. of Otorhinolaryngology, Dept. of Surgery, Univ. of Arizona Health Sciences Center, 1501 N. Campbell Ave., Tucson, AZ 85724.

Definition

In discussing this problem, we initially must define: 1) elective radical neck dissection, 2) therapeutic radical neck dissection, and 3) secondary or salvage radical neck dissection.

A RND is considered *elective* if there is no palpable cervical adenopathy but a known head and neck primary lesion.

A RND is considered *therapeutic* if cervical adenopathy is palpable and co-existent with a known head and neck primary lesion.

A RND is considered *secondary* or *for salvage* in patients in whom the primary tumor is thought to be controlled but in whom clinical cervical adenopathy arises.

Technique

Although the major objective of this paper is not one of technical aspects of RND, it would be well to point out the "standard" RND and variations. There are numerous incisions which can be utilized; figure 1 shows the three which we most commonly use. The basic principle behind any incision in this case is to attempt to give adequate exposure, to keep incisions from overlying the carotid sheath, and to provide optimal cosmetic results. Thus, for most of our cases (especially those where intra-oral exposure is necessary), we utilize the Conley incision. For a laryngectomy with a RND, the Utility (Lahey) incision is used. Occasionally, the MacFee is performed if cosmesis is of extreme importance.

After the skin flaps have been designed, the flaps are elevated deep to the platysma muscle if possible. This gives a better blood supply to the skin and, thus, lessens necrosis. Superiorly, the ramus mandibularis branch of the facial nerve is preserved if possible.

Inferiorly, the sternocleidomastoid muscle is transected 1 cm. above the clavicle. The internal jugular vein is freed circumferentially, ligated, and transected after the surgeon makes certain that the vagus nerve is identified. The supraclavicular fat pad is removed, with care being taken to preserve the phrenic nerve and brachial plexus which lie deep to the prevertebral fat pad. On the left side, one must attempt to preserve the thoracic duct or, if it is damaged, to locate and ligate all branches, using an operating microscope if necessary.

The dissection is carried to the anterior edge of the trapezius (the posterior margin of the dissection). Then the dissection is carried superiorly to the upper end of the sternocleidomastoid muscle. It is during the posterior portion that the spinal accessory nerve is usually sacrificed. The internal jugular vein is again ligated and transected near the posterior belly of the digastric muscle, and the submandibular triangle with its contents is removed with care being taken to preserve the hypoglosssal and lingual nerves. The submental triangle contents are removed, completing the dissection. The wound is irrigated, two under-the-flap suction drains are inserted (one posterior and one anterior to the carotid artery), a dermis graft from the thigh is placed over the carotid artery if the patient has been irradiated or if the oral cavity or hypopharynx is opened, and the wound is closed in two layers. No external dressing is applied.

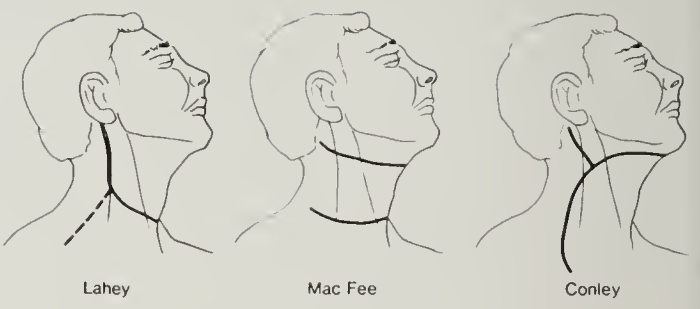

Figure 1
Three commonly used incisions for radical neck dissection in head and neck carcinoma.

Variations of the RND (modified RND) have been described including preservation of the XI nerve, grafting of the XI nerve if sacrifice is necessary, perservation of the internal jugular vein, and limited supra-hyoid dissections. Unless there is an unusual extenuating circumstance, we rarely preserve the XI nerve or the internal jugular vein and utilize the supra-hyoid dissection for small anterior tongue or floor of mouth lesions.

Elective RND

Decisions concerning RND must be predicated on the accuracy of the clinician's evaluation of cervical metastasis. Sako et al,[1] in 235 patients, felt that the ability to palpate a node depends upon its consistency and location. In a superficial area, such as the submental or submaxillary regions, the lower limit of palpability is 0.5 cm., while in deeper areas it is thought to be 1.0 cm. The accuracy of diagnosis was 72.3% for palpable nodes and 72.4% for nonpalpable nodes. Thus, in making a decision concerning an elective RND, one must consider the possibility of clinical errors in palpation in approximately 25% of the cases.

It is in this group of patients that the main controversy lies. In an extensive review of the literature, Nahum et al[2] found numerous articles in the recent literature which question the value of prophylactic or elective RND. Basically, the case against an elective RND is derived from the data obtained from aggressive radiation therapy centers where figures are quoted of only 5% failure.[3] However, Lee and Krause,[4] in an in-depth review of 1,584 cases of primary tumors of the oral cavity, hypopharynx, oropharynx, and larynx found a much higher 3-year determinate survival rate in surgical (83%) or combined therapy (93%) patients than in those treated by radiation (65%) alone. For 5-year determinate survival rates, surgery had 79%, combined therapy 93%, and radiation, 63%.

Some authors[5,6] feel that elective neck dissection will benefit only approximately 5% of patients in terms of survival statistics. It is argued that, in view of a surgical mortality of 3%, it is unfair to subject 95% of the population to this mortality figure unnecessarily. Lee and Krause not only point out the marked differences in survival in their series, but also answer this point about surgical mortality. They feel that if a patient has a 20% or greater chance of occult cervical metastasis, then a treatment plan involving an elective radical neck dissection is warranted.

Therapeutic RND

There is little disagreement on the efficacy of the therapeutic RND. Also, in most series, it appears that a combination of surgery and irradiation (given either pre- or post-operatively) is superior only to surgery or irradiation alone. The series of Lee and Krause varied slightly in that the 5-year determinate survival rates were 60% for combined treatment, 60% for surgery alone, and 32% for radiation alone.[7]

Questions have been raised by Santos et al[8] concerning the role of RND in lesions with fixed adenopathy. In their small series of 12 patients, nine died of disease within 3 years, one died of an unrelated cause but without disease, and two were cured. However, it should be emphasized that these patients received varying doses of radiation (3100 rads on a 2000 kv machine to 7000 rads cobalt 60) preoperatively.

We feel that surgical reduction of tumor size followed by high (6000-7000 rads) doses of irradiation for residual microscopic disease will improve these figures.

Salvage RND

Fayos and Lampe[9] reported on irradiation of 320 patients with squamous cell carcinoma of the tonsillar region, floor of the mouth, anterior two thirds of the tongue, and lower alveolus. In the N_0 group, 43 of 206 (20.8%) developed ipsilateral neck metastasis. Of these, 32 underwent RND with salvage rate of 50% (16/32). This coincides with the 53% 5-year survival in salvage procedures reported by Lee and Krause.[4]

RND After Radiation Therapy

With the increased utilization of radiation therapy, this topic is becoming extremely important. As Donald points out, ". . .many reports by radiotherapists, which extol the virtues of surgical salvage in patients with irradiation failure. They claim that an inordinately high incidence of complications in these patients is not forthcoming".[10] He proceeds to point out that this is in contrast to reports by Joseph and Shumrick,[11] Baker,[12] Habel,[13] and Trible[14] where the opposite view is presented. Here, in all fairness, one should emphasize that the latter three references were from 1963, 1965, and 1957, respectively, and that the radiation techniques have changed. However, in Donald's own series, 45% of his salvage cases had major complications (carotid artery blowout, fistulae persisting 10 days or longer, major loss of skin flap, wound separation of 6 cm. or greater, or osteoradionecrosis). Joseph and Shaumrick had a 77% wound dehiscence rate and 50% incidence of carotid artery rupture (this was before dermal graft protection of the carotid). Thus, the problems encountered in salvage after radiation are significant, especially when one is dealing with only 50% salvage statistics.

Goodwin and Chandler[15] reviewed 360 radical neck dissections in an attempt to determine the efficacy of "elective" neck dissections after a patient had received "therapeutic" (5000 rads or more) doses of radiation therapy to the neck prior to surgery. In original N_0 or N_1 stages with *no* palpable nodes prior to "salvage" surgery following irradiation, 18% (4/22) had residual disease. In original N or N disease with *no* palpable nodes prior to "salvage" surgery, residual occult disease was found in 50% (5/10) patients. Thus, they feel that if a patient has N or N disease in the original staging, the high incidence of residual disease following irradiation justifies "elective" RND even when no nodes are palpable.

Complications of Radical Neck Dissection

Although we shall not dwell on complications (the topic will be covered in a later article), we would be remiss in omitting the topic completely. Skin flap elevation due to bleeding from the wound occurs occasionally and thus under-flap suction drains are utilized. Infection, especially when the oro- or hypopharynx is opened, is a concern and, consequently, prophylactic systemic antibiotics are useful. Skin necrosis, salivary fistulae, chylous fistulae, carotid artery rupture, and reduced ipsilateral upper extremity motion (due to spinal accessory sacrifice) are all potential problems. However, the incidence of these complications has been significantly reduced with better design of skin flaps, dermis graft or muscle flap coverage of the carotid artery, and early, aggressive postoperative physical therapy.

All too frequently, age is give as a contraindication to surgery of head and neck malignancies. Excellent data to the contrary are now being accumulated. McGuirt et al[16] found that of 714 RND's performed at the University of Iowa Hospitals from 1963-1973, 162 were in patients over 70 years of age. The incidence of major complications (carotid blow-out, fistulae, operative mortality) was 17.9% in this group as compared to 20.5% for those less than 70 years of age. Medical complications (cardiovascular, pulmonary, gastrointestinal bleeding, renal insufficiency) were greater in the older patients (19.8%) than in the younger patients. However, in view of these accurate data, he concluded that conservatism in the elderly patient with head and neck cancer may actually be a "radical" poor selection.

Discussion

In view of the above data, we attempt to adjust our management of the patient with a head and neck malignancy to meet the individual case. It is essential that the surgeon maintain a close working relationship with the radiation and medical oncologists. In general, because of the findings concerning salvage surgery and the increased incidence of complications after irradiation, we would perform a radical neck dissection if the chance of metastatic disease is 20% or more and then would utilize postoperative irradiation. We do not feel that nodal fixation is an absolute contraindication to resection, since reduction of tumor size may allow the radiation oncologist to manage any residual, microscopic disease.

Summary

In summary, an attempt has been made to present an overview of the controversies surrounding the modality of the radical neck dissection (RND) in the management of the patient with head and neck carcinoma. Varying viewpoints have been brought out, as well as the techniques of the procedure, the potential risks, and the potential benefit to the patient. The RND continues to be efficacious in all age groups and is indeed *not* truly a "radical" or "mutilating" procedure *in and of itself.*

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Seminars in
Endocrinology and
Metabolism

Pituitary Disorders: The Role of Prolactin

Stephen E. Dippe, M.D., FACP
M. B. Block, M.D., FACP

Editor:
Stephen E. Dippe, M.D

Until recently most pituitary tumors were diagnosed when skull radiographs showed an enlarged sella turcica or when symptoms of reduced peripheral vision occured or obvious signs of hormone excess or deficiency were present. Today, however, we are discovering pituitary gland abnormalities earlier due to accurate measurements of plasma pituitary hormones.

The development of the prolactin radioimmunoassay has greatly expanded our knowledge of this hormone and its relationship to several clinical disorders. A brief review of the most important of these relationships follows.

From: Scottsdale Memorial Hospital, Family Practice Center, 7301 East 4th Street, Scottsdale, AZ 85251. (Dr. Dippe). Endocrinology Associates, PA, 3411 North 5th Ave., Phoenix, AZ 85013 (Dr. Block).

It was not until the early 1970's that we even felt onfident that the hormone existed by itself. Prolactin is n unusual hormone in that its regulation is different rom other hormones of the anterior pituitary gland. Vhile most pituatary hormones are stimulated by ormones from the hypothalamus, prolactin is ontrolled, in addition, by an inhibitory hormone called uccinctly, Prolactin Inhibitory Hormone (PIH). Dopamine is known to stimulate PIH and in addition it may even be a part of prolactin's releasing hormone.[1] The releasing hormone for prolactin may be Thyrotropin Releasing Hormone (TRH). People with hypothyroidism have elevations of TRH and may indeed have galactorrhea from the elevated serum prolactin levels. These people do not have a prolactin-secreting tumor and simply replacing the patient with thyroid hormone will reduce the prolactin level and the galactorrhea that sometimes occurs. Whether TRH truly has a normal physiological action on prolactin secretion remains obscure. For example, mothers who are breast-feeding do not seem to stimulate TRH yet the level of prolactin is elevated.

A number of physiological stimuli increase prolactin. The most significant stimulant is nursing but also stress, sleep, and occasionally strenuous exercise can increase prolactin.[2] During pregnancy the prolactin level rises. A number of drugs affect the prolactin level. For example, phenothiazines such as Chlopomazine, Reserpine and Methyldopa can increase the prolactin level. Longterm use of estrogens has been shown to increase the level of prolactin. L-dopa and apomorphine inhibits the release of prolactin. Ergot derivatives have recently been shown to be effective in inhibiting prolactin release. The most promising drug in this category is bromergocryptine.

The measurement of prolactin is most useful in patients with amenorrhea and/or galactorrhea syndromes. High levels of prolactin are now known to inhibit menses and cause infertility in women and impotency and infertility in men. How this is done is still not known but prolactin may act on the hypothalamus to inhibit the gonadotrophin releasing hormone or may inhibit the gonadal response to FSH and LH. The important thing to realize is that hyperprolactinemia can exist with amenorrhea and/or galactorrhea and that restoration of menses and secession of galactorrhea has occurred following treatment of bromergocryptine. In the older literature, there was a multitude of syndromes attached to these conditions such as Forbes-Albright Syndrome, Chiari-Frommel Syndrome and Del Castillo Syndrome. These eponyms should be discarded. Some patients with the amenorrhea-galactorrhea syndrome will have demonstrable pituitary enlargement and others have nothing to suggest a pituitary tumor with the exception of having an elevated level of prolactin.

A review of 235 patients with galactorrhea revealed that the largest single group (76 cases), were women with galactorrhea and without amenorrhea.[3] Of these, 86% had normal prolactin levels. In contrast, only two of twenty patients with galactorrhea and amenorrhea were the prolactin levels normal and of the eleven patients treated with bromergocryptine, seven had disappearance of galactorrhea, eight had resumption of menses and two became pregnant. The highest prolactin levels were found in patients with pituitary tumors.

It should be remembered that not all female patients with hyperprolactinemia result in galactorrhea and in men it's rare. A report by Bohnet, et al, showed that eight patients with hyperprolactinemia had amenorrhea but never developed galactorrhea.

Management of prolactin levels is useful in diagnosing the cause of an enlarged sella turcica. In men, for example, we frequently do not find galactorrhea with elevated serum prolactin levels. However, infertile and impotent men, may have an elevated prolaction level which may suggest a pituitary lesion. Once surgery or radiation has been performed on the pituitary lesion, prolactin levels can be used to follow the postoperative course to see if there is any reoccurrence of the tumor.

Therapy for pituitary tumors has also changed in recent years. External radiation has been around for quite some time and is for the most part safe. It does, however, take a number of months to achieve the maximum effect of the treatment and sometimes hypopituitarism occurs. Because of the delay before results are seen, external radiation may not be appropriate for those who have visual impairment or are desirous of being fertile afterwards. A "non" approach Transsphenoidal hypophysectomy (see *Arizona Medicine* Vol. 36, No. 10, 1979 p.756) can be used quite effectively to remove pituiatry adenomas and has become a major means of treating intrasellar lesions. Maintaining normal pituitary function postoperatively is usually possible. Finally, bromergocryptine has been shown to be effective in reducing prolactin levels and even in reducing the size of prolactinomea in carefully selected patients, but longterm follow-up of large number of patients is not yet available.

Supported in part by the Edyth Bush Charitable Foundation, Inc.

Special Article

Abortion Reporting

Mary Rita Stark

In July 1976, the Arizona Department of Health Services began in Induced Termination of Pregnancy Reporting Program aimed at obtaining statistical information on abortional acts performed in the State. This is in accordance with Section R9-19-302D of the Rules and Regulations of the Department of Health Services, as authorized by ARS 36-344, requiring the reporting of abortional acts performed in hospitals, outpatient treatment centers, and physicians' offices.

During 1978 a total of 6,833 termination reports were recorded by the Department of Health Services. This figure compares to 6,130 terminations recorded in 1977. However, according to Roy Walker, Chief of the Bureau of Vital Records and Information Services, these figures represent only the actual number of reports received, not actual number of abortions performed.

"We wish to remind physicians that they are required to submit Induced Termination of Pregnancy reports to this office", Walker stated. He emphasized that the responsibility for filing these reports rests with the attending physician, not the hospital or clinic where the procedure was performed.

Forms for reporting induced termination of pregnancy are available from the Research and Statistical Analysis Section of the Bureau of Vital Records and Information Services. They are for gathering statistical information only; no personal data is recorded.

The recorded information includes age, race, marital status and number of years of education of the mother, as well as the reason for termination, procedure used and any major, immediate complications. Walker explained that ADHS is the central repository for this data in Arizona. The data is available to the public, but most requests come from health planning and family planning agencies.

For additional information, or to obtain reporting forms, contact: Arizona Department of Health Services, Research and Statistical Analysis Section, 1740 West Adams Street, Room 103, Phoenix, Arizona 85007. Telephone: (602) 255-1068.

From: Arizona Department of Health Services, 1740 West Adams Street, Phoenix, AZ 85007

State of the Art

Atypical Mycobacteria

Robert B. Cobb, M.D.
Russell Dodge, M.D.

Editor:
Eric H. Prosnitz, M.D.

Introduction

During the past several decades the prevalence of tuberculosis has steadily declined in this country. Human disease caused by other members of the Mycobacterium genus, however, has continued at a constant rate throughout this period.[1] As a result, the atypical mycobacteria—that is, those organisms producing mycobacterial infections other than tuberculosis—are responsible for an increasing proportion of the morbidity and mortality of the mycobacterial diseases.

Until approximately one decade ago, the management of patients with all forms of mycobacterial disease was the concern of medical personnel in specialized tuberculosis hospitals. Now, with these patients being cared for in general medical outpatient clinics and hospitals, they are managed by physicians not specializing in pulmonary or infectious disease.

These two developments—the increasing clinical importance of the atypical mycobacteria and the return of patients with nearly all forms of mycobacterial infection to the care of general practitioners and internists—make it essential that all clinicians become familiar with the atypical mycobacterial diseases.

From: Division of Respiratory Sciences, U of A Health Sciences Center, Tucson, AZ 85724 (Dr. Cobb) and Veterans Administration Hospital, Tucson, AZ 85723 (Dr. Dodge). Dr. Cobb supported by National Research Service Award HL07243.

istorical Background

The existence of various types of mycobacterial has een known since the early days of mycobacteriology. In he 1880's, shortly after Robert Koch announced his scovery of the human tubercle bacillus, numerous acteriologists described similar acid-fast bacilli isolated om non-human sources. Moeller, in 1899, reported ecovery from humans of organisms very similar to hose he had earlier isolated from the environment. rom his own nasal mucus he was able to isolate acid-ast organisms on three separate occasions. This work stablished the possibility of human colonization, but Moeller and his colleagues did not believe that these rganisms were pathological for humans, since they were not virulent for the guinea pig. The development f improved culture techniques and their use as a iagnostic tool resulted in a change in attitude. Strains virulent for guinea pugs were repeatedly cultured from putum and resected lung tissue of diseased humans, nd in the face of failure to find other disease agents, hese cultures constituted strong evidence for the pathogenic nature of these mycobacteria. Once it was ccepted that these organisms caused disease in humans, nformation concerning the various species which were pathogenic for man rapidly accumulated. For example, M. Kansasii was isolated from two patients at necropspy by Buhler & Pollak in 1953; and in 1957, the Battey bacillus was discovered in a patient's sputum at the Battey State Tuberculosis Hospital in Rome, Georgia. New species are continually being isolated, and both these new species and older ones are being casually related to human illness.

The study of the atypical mycobacteria has been standardized by a system of classification developed by Timpe and Runyon in 1954.[2] A modified version of this classification is shown in Figure 1. The importance of this classification lies in its division of slowly growing from rapidly growing organisms, and the further division of the slow growing organisms according to the very simple and readily understood characteristics of culture pigmentation.

Epidemiology

The atypical mycobacteria are ubiquitous in man's environoment. Wolinsky and Ryenearson isolated M. fortuitum, M. avium and M. intracellulare from soil samples collected in various parts of the United States.[3] M. intracellulare has also been isolated from the spray and foam of ocean water.[4] Tap water has yielded cultures of M. xenopi, M. gordonae and M. Kansasii. Humans are not case sources of clinical disease, and the atypical mycobacterial infections are not transmitted from person to person. Although there are reports of family aggregation of cases, these few instances probably represent an acquisition of infection from a common environmental source. At the present time, it is felt that patients with either active mycobacteriosis or persistently positive sputa are not infectious and need not be isolated.

Geography plays a part in the occurence of clinical disease secondary to these organisms. Group III disease prevails throughout the Southeastern United States, while Group I seems to predominate in New Orleans, Houston, Dallas, and especially Chicago, where 8 to 10 percent of all new admissions to tuberculosis services may be secondary to this group of organisms. Disease produced by the Group II organisms is rare and has occurred most freuqently around the Great Lakes. The importance of atypicals in Arizona is indicated by incidence figures from the Veterans Administration Medical Center in Tucson, During the period 1976 through 1978, there were a total of 124 new patients with disease caused by mycobacteria. Of these cases, M. tuberculosis constituted 63.7 %, while M. intracellulare and M. Kansasii represented 19.3 % and 8.1 % respectively.

Pathogenicity

In the majority of cases, these organisms, when isolated from sputa or gastric washings, lack pathologic significance. In one study, for example, atypical mycobacteria were isolated from the sputum or saliva of a surprisingly high proportion of a healthy adult population in Georgia.[5] In another study at the Ohio Tuberculosis Hospital, gastric washings of health employees also contained atypical organisms.[6] The interpretation of a positive culture should take into consideration the possibility of environmental contamination and the known pathogenic potential of the recovered organisms. Usually a single, nonrepeatable positive sputum culture with scanty growth is of no clinical significance. However, the repeated isolation of an organism in a patient with a compatible clinical picture should be considered significant.

An interesting aspect of these infections is that they usually occur in patients who have underlying lung disease. The most common predisposing conditions include previous tuberculosis, chronic bronchitis, emphysema, bronchiectasis, pneumoconiosis and chronic aspiration from esophageal disease. The association of silicosis and atypical mycobacteriosis has been reviewed extensively. In a study of silicotic sandblasters, Bailey et al found that M. kansasii or M. intracellulare caused 41% of the complicating mycobacterial infections.[7] This study appears to be the clinical counterpart of the experiment described by Gardner in which guinea pigs dusted with silica often developed overwhelming infection when exposed to atypical organisms that would not otherwise cause disease.[8] It must be emphasized, however, that infection also does occur in those persons without apparent lung disease or dificiency of cellular immunity.

Clinical Aspects

Chronic pulmonary disease is the most important clinical problem associated with the atypical mycobacteria, and of the atypicals, M. kansasii and M. intra cellulare are the most common pulmonary pathogens.[9] M. scrofulaceum, M. marinum, M.

fortuitum and M. chelonei are rarely the cause of human disease. M. xenopi, occasionally reported as a source of infection in North America, is commonly a source in Europe. Mycobacteriosis appears to be a disease of white males over the age of 40 in whom the lesions are both microscopically and macroscopically characteristic of those produced by M. tuberculosis.

Differential skin testing has been used extensively in the past in the diagnosis of mycobacterial disease. Antigens have been prepared to M. tuberculosis (PPD-S), M. kansasii (PPD-Y), M. intracellulare (PPD-B) and M. scrofulaceum (PPD-G). But because many surfact antigens are shared among mycobacterial species, it is not surprising that cross reactions interfere with proper interpretation for diagnostic purposes. Therefore, although a useful epidemiologic tool, differential skin testing is not helpful in usual clinical practice, and the only reliable method for differentiation between tuberculosis and mycobacteriosis is laboratory indentification.

Mycobacterium kansasii

Studies of the antigens of M. kansasii place it closer to the tubercle bacillus than any of the other atypical mycobacteria. Disease due to this organism has been reported from all parts of the world. In some geographic areas of the United States, this organism may constitute up to 20 percent of mycobacterial disease. It usually occurs in adults between the ages of 40 and 60, with males predominate in a 3 to 1 ratio. Occupation has no definite relationship to occurrence of the disease, except for the association with silicosis as seen with sandblasters. Diabetes mellitus is not commonly associated with this organism, nor is alcoholism seen as often as in patients with tuberculosis. Those affected are usually urban dwellers.

Pulmonary symptoms are similar to those of tuberculosis, but usually milder than expected, considering the amount of disease revealed by chest film. Even in moderately advanced cases, the patients frequently have few symptoms. Fever is often absent, with less than 5% reporting abrupt onset of high, spiking fever and night sweats. Hemoptysis is fairly common and often is the reason for seeking medical attention. Physical findings and laboratory examination are similar to those of tuberculosis. Progression of M. kansasii infection is unpredictable, but Francis has shown that the untreated disease nearly always worsens with continuing destruction of lung tissue over many months or years.[10] Characteristically, however, pulmonary infection with this organism does not involve extrapulmonary spread except in immuno-compromised hosts, lending support to the observation that M. kansasii is less invasive than M. tuberculosis.

The response of M. kansasii infection to drug therapy is generally good because of its susceptibility to rifampin and only slight resistance to the other antituberculosis drugs. Several studies indicate a 50 to 80 percent rate of sputum conversion to negative after six month of therapy.[11, 12] Harris and associates have presented t results of their experience with 58 patients with kansasii infection.[13] In the group receiving their init course of chemotherapy, 34 of 38 patients beca culture negative. This group was followed for periods 12 to 35 months with no bacteriological relapse. Har found that those treated with rifampin had an earli and more complete sputum conversion. Even mo striking were the results in 24 patients undergoi retreatment: All 16 in the rifampin group convert their sputum culture to negative within 9 months, b only 3 of 8 patients treated without rifampin becan bacteriologically negative after 24 months. Only one the 58 patients required surgical resection because persistently positive sputum and open cavities after months of chemotherapy; and since surgery, he h remained bacteriologically negative on chemotherap Most investigators recommend triple therapy wit rifampin, isoniazid and ethambutol for a period of years. The need for surgical intervention appears to b decreasing as good results with long-term combine chemotherapy continue to be observed.

Mycobacterium avium - intracellulare complex

The majority of cases of illness secondary to thes Group III organisms involves white males usually ove the age of 50. Patients are likely to be from lowe socioeconomic groups and to have lived in rural areas o the Southeastern United States.

The disease is chronic and insidious in its development. Symptoms often have been present fo years. The most common complaints are cough weakness, and weight loss. Fever is infrequent an usually low-grade. As with M. kansasii, the rate of progression of untreated disease is unpredictable, but usually is slower than that of untreated tuberculosis. Often the major medical problem and ultimate cause of death is not the mycobacteriosis, but the underlying lung disease. Occasionally patients will remain clinically stable for years with untreated active infection.

This highly variable course in patients who often have extensive underlying lung abnormalities makes decisions about therapy very difficult. Because of naturally occurring drug resistance, disease associated with M. intracellulare responds less well to drug therapy than M. kansasii. Most strains are resistant to all of the antituberculosis drugs. The poor results of therapy were summarized by Yeager et al in their study of 45 patients.[14] Despite the use of 4 or more drugs in approximately one half of the patients, and adjunctive surgical treatment in 42 percent of the patients, less than one half of the group was alive with inactive or quiescent disease 5 years later. Even in those with sputum conversion, the cumulative rate of relapse was 20 percent after 5 years of treatment and observation. In a more recent study of 100 patients treated with multiple drug regimens including rifampin, sustained conversion of sputum was achieved in only 45 percent of 82 patients.[15] Eighteen of the 82 underwent lobectomy after a 6 month trial of drugs; and even with continued chemotherapy, only 11 of these 18 achieved lasting stability. In another study at the National Jewish Hospital, Davidson used an intensive regime of at least 5 drugs and resectional surgery when indicated.[16] Successful treatment was obtained in 77 percent of 93 patients, and of these, seven percent were found to have relapsed on follow-up.

Although many drug regimens have been recommended, the usual therapy in a previously untreated patient would consist of one parenterally administered drug (usually streptomycin), and four or five orally administered drugs such as rifampin, isoniazid, ethambutal, cycloserine or ethionamide. This regimen should be continued until the patient has been culture negative for one year; then three or four of these drugs should be continued for a total of two years after culture conversion.

Often therapy is limited by drug toxicity, making hospitalization advisable for initiation of the medications. After discharge, patients should be seen frequently by a physician. If toxicity necessitates discontinuation of a drug, another should be substituted, especially early in the treatment course. Usually successfully treated patients show a clinical response within three months; some may require up to six months to become consistently culture negative.

Surgical resection may be useful in patients with localized M. intracellulare infection who are good operative candidates.[17-19]. Occasionally, local resection is indicated as the initial therapeutic procedure, but should only be performed following preoperative chemotherapy. Usually, however, the patients who undergo surgery are those with disease which has progressed in the face of chemotheraphy, but is still localized.

Other Atypical Mycobacteria

Because of the small number of patients involved, experience in management of the less frequently encountered mycobacteria is limited. The pulmonary pathogens M. scrofulaceum, M. chelonei and M. fortitum are very resistant to antituberculous drugs in vitro. Usually treatment is initiated with a multiple-drug therapy like that for M. intracellulare. Because in vitro studies occasionally show the organism to be sensitive to erythromycin, tetracycline, sulfa or the cephalosporins, some investigators include one or more of these drugs in the regimen. Recent studies have indicated that amikacin may be useful in treating infection caused by M. fortitum and M. chelonei.[20] M. xenopi is more susceptible to drug therapy than the above mentioned organisms, and currently the recommended regimen is the same as for M. kansasii. M. marinum, which causes localized cutaneous lesions, can be treated by surgical excision; drug treatment is not indicated unless there is extensive, deep involvement.

In summary, the atypical mycobacteria, especially M. kansasii and M. aviumintracellulare, are an increasingly important group of pathogenic bacteria. Clinicians in a primary care setting, while appreciating how often isolation of atypicals is of no clincial significance, should be prepared to diagnose and treat patients with disease secondary to these organisms. Treatment results of M. kansasii have been encouraging, but the management of M. intracellulare may be extremely difficult.

Because of the toxic drug regimens and the difficult surgical treatments frequently employed, patients unresponsive to initial therapy may need referral to specialized centers.

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Arizona Medicine

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Heart Transplantation:

Human Experimentation or Therapeutic Intervention?

During the middle 1950's and early 1960's the current surgical techniques for heart transplantation were developed in the Stanford University surgical laboratory by Shumway and his early residents. The necessary and initial contribution of that laboratory was description of a simplified surgical technique. For the first time, it became practical to implant a heart in the orthotopic, or normal anatomic position within a reasonable period of time (30-45 minutes). These investigators also described and tested various treatment regimens. Autotransplantation, or removal and replacement of the animal's own heart, eliminated the problem of rejection and provided them with a model for studying the long-term effects of cardiac denervation.

Although the Stanford team, after more than ten years of research, had declared it was ready to perform human heart transplantation in mid-1967, the first successful transplant was performed in the Union of South Africa in December, 1967. An epidemic of similar operations occurred around the world, but the results were poor and most surgeons and cardiologists lost enthusiasm. The Stanford team, however, continued to enlarge its experience and learn more about cardiac transplantation. Improved selection criteria were evolved which required candidates to be age 50 years or less with no diabetes mellitus, pulmonary hypertension, pulmonary infarction, or active infection. Combining the very sensitive technique of following serial electrocardiographic voltage with the very specific technique of percutaneous transvenous right ventricular biopspy, the diagnosis of rejection could be proved with certainty. Patients were treated for rejection only when it definitely occurred and over-treatment was reduced. Rabbit antithymocyte globulin was added to the usual acute medications, prednisone and azathioprine. After the addition of the anti-platelet aggregation agent dipyridamole to long-term medical therapy, the incidence of accelerated arteriosclerosis in heart grafts w reduced by one-half. Finall retransplantation or second transplant tion was established as an alternative f patients whose initial heart transplan failed due to rejection or arteriosclerosi

The results of these improvemen were dramatic. A group of patients wa identified who without hear transplantation had a 100% mortality rat within one year as compared to 60-70 survival rate for one year wit transplantation. This survival rate wa equal to the kidney graft survival rat following cadaver kidney transplanta tion. The patient attrition rate afte transplantation has been 5% per yea resulting in a five year survival rate of 50 55% for recent cohorts of patients. Thes statistics have remained essentiall unchanged for the last five years in th Stanford cardiac transplantatio program and have been reproduced at th Medical College of Virginia.

Such excellent statistics have stimulated programs in other centers including the University of Arizona. The number of heart transplants in the world thus is increasing each year reaching 40 to 50 per year in the last two years Rehabilitation of patients after heart transplantation has been critically evaluated and 90 percent of patients who survive one year or more reach full working status or full rehabilitation.

The cost of cardiac transplantation has been high. The average cost of cardiac transplantation for the initial hospitalization at the University of Arizona has been $23,500. This cost undoubtedly will be significantly reduced. The average cost of a kidney transplantation is currently in the $20,000 range.

Cardiac transplantation is no more experimental than kidney transplantation. It is simply less commonly done. A demanding procedure, it requires a great deal of physician time and interest, a basic background of factual material, and a mastery of several techniques. In a spirit of cautious optimism and with a desire to make the procedure less expensive and more practical, we are proceeding with the program at the Arizona Health Sciences Center.

Jack G. Copeland, M.D.
Associate Professor of Surgery
Louis J. Kettel, M.D., Dean
College of Medicine

Auxiliary Highlights

Auxiliary Monitoring HSAs; Developing PR Program

Auxiliary members in Mohave, Yuma, and Pima Counties have begun monitoring Health System Agency meetings in their areas and Maricopa County Auxiliary is recruiting Auxilians to serve as observers at CAHSA committee meetings.

State Legislation Chairman Ruth Ogden, at ArMA's request, has briefed Auxiliary county presidents and legislation chairmen on HSA function and committee structure. She has suggested they assign Auxiliary members to act regularly as observers at meetings of specific committees. In that way, the observer will become more familiar with committee members and their work. A form has been designed which Auxilians can use to report on each meeting they cover. Copies of the report will go to the county society and to ArMA for their information.

Mrs. Ogden is preparing an audio-visual slide show explaining HSA structure, which can be used in the various counties. It is based on a similar program used by the California Medical Association Auxiliary and is yet another example of the increasing cooperation between the medical auxiliaries of the western states.

Mrs. Robert Evert and Mrs. Eugene Eldredge, president and president-elect of the California Medical Association Auxiliary, came to Phoenix for the Arizona Auxiliary's Fall Conference in September. Earlier, Mrs. David Newman, Mrs. George Hoffmann, and Mrs. John Clymer attended California's Fall Conference . . .four days packed with lectures and workshops covering everything from legislation to public relations. We were especially interested in the TV training California Auxiliary offers doctors and Auxilians.

New PR Projects

Arizona's Auxiliary has embarked on an ambitious public relations program. Membership chairman Janet Hoffmann, public relations chairman Jeanne Davis, and newsletter editor Barbara Dubin have prepared a questionnaire, "Who Are We?", which will be sent to all spouses of ArMA physicians. Mrs. Dubin will use it to develop an Auxiliary profile which will be featured in the winter issue of the newsletter. Information from the questionnaire will be helpful in planning future activities of the Auxiliary.

Photographer Annette Schneider of the PR committee is working on a photo montage showing Auxiliary members at home and in the community to display at Convention. Kay Dries will use her photography and graphic skills to produce a color slide show, "Here is Your Auxiliary". Judy Davis of Prescott, second vice president of the Auxiliary and active in theater, is developing a "Traveling Medicine Show" which will tell Arizona doctors' story to school children.

Mrs. John T. Clymer
ArMA Auxiliary President

Kennedy's New Health Proposal:

More Slogan Than Reality

As their National Health Insurance plan of the year, Senator Ted Kennedy and his helpmates have built a bright-eyed new statue with the same old clay.

The new "Health Care for All Americans" bill differs from his old "Health Security" bill in various particulars but not in basic materials.

Again the public would save on health bills by paying substantially more in tax bills. By the proponents' own estimates, the new federal costs would be $30 billion in 1983, the year targeted for implementation. And as we know, the costs of federal programs—especially social ones—have a facility for growing.

Again there would be the heavy hand of government intervention and regulation.

But instead of being squeezed under, as in the Health Security bill, it appears private health insurers would be allowed a role of subordination. Government would regulate them and preside over negotiations on their premiums and on physician and hospital payments, and no company would be "at risk" in the true insurance sense.

In the spirit of the old bill, there would be a built-in stress and strain between health care promises and economic prohibitions.

On the one hand, the measure would guarantee comprehensive health insurance coverage to every American at all times, amplify Medicare coverage (with no deductibles or co-insurance), reform Medicaid (eliminating the means test), and so forth.

On the other hand, the program would be subject to a national budget, whereby health expenditures would be strictly limited by state and regional allocations. Providers would have to furnish all services within the limit.

If funds run out before the end of a budget period, would providers be expected to subsidize health services? Would budget restraints and negotiation of insurance premiums and provider payments invite government rationing of care?

Those are among the grim but realistic questions to be raised as Mr. Kennedy and his supporters try to supply all the answers.

Certainly we all want answers—sound answers—to present gaps and shortcomings in health protection. In the words of AMA Executive Vice President, James H. Sammons, M.D., "The AMA continues to believe that consumer choice, private insurance, and limited government regulation should be at the heart of our health care system."

"Medicine again faces a renewed responsibility to speak out on the major legislation proposals dealing with health care and to share our concerns with the Congress and our fellow citizens."

American Medical Association

Correspondence

Editor—

Consider the "Jarrett Decision" (no connection), that has far reaching implications, and promises to keep lawyers busy for sometime to come. It seems that Dr. Jarrett examined a boy who had been injured in a hit-and-run accident, for the purpose of determining residual injury, prognosis and disability. The insurance company of the boy's father was responsible under the "uninsured motorist" clause. The boy by and through his father, sued the insurance company and obtained a favorable settlement. He then sued Dr. Jarrett on the grounds that his report of the residual disability and prognosis were unduly conservative and allegedly resulted in an inadequate settlement which required litigation costs and attorney fees in filing a prior action against the insurer. The damages against the doctor were small since the insurer had paid on the basis of the court's award, so a second count alleged that the doctor's conduct was "intentional, willful and fradulent". The trial court dismissed the suit on the ground that the insurance company had not been named in the suit also, although by now they had settled. The appeals court, however, *reversed* the trial court's dismissal of the action and presumably the case is awaiting retrial. [Civ. No. 14877. Third Dist. Oct. 3, 1977 (Cal.)]

Boggles the mind, doesn't it?

Paul B. Jarrett, M.D.

Editor—

I have read Dr. Dermont Melick's letter, *Arizona Medicine* Vol. 36, No. 10 p. 708; the following are my additional comments.

As president of the Medical Staff at St. Luke's Hosptial, I have been overwhelmed with the negative response on the part of physicians to the present CPR course. Doctors, as a group, are totally dissatisfied with the way these courses are being run in the various hospitals. The have no interest in listening to a group of nurses berate them with comments such as, "We're getting tired of helping you bury or resusitate your mistakes."

Physicians are not interested in learning paramedic procedures. They want something with more depth and clinical application. I know what your comment will be to this, but let's see if we can't make this more palatable to physicians. Above all, let's get the nurses out of it.

Because of all this negative response. I'm going to ask that the St. Luke's staff take the course on a voluntary rather than mandatory basis. We hope that St. Joseph's Hospital and Good Samaritan will follow suit.

Howard H. Johnston, M.D.

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. . . Bentyl produced definite relaxation in 8 of 10 patients. The sodium chloride produced relaxation in only 3 of 10. No side effects occurred in either group of patients.

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†See Warnings, Precautions and Adverse Reactions.

See following page for prescribing information.

Reference:
King, J.C. and Starkman, N.M.: Evaluation of an antispasmodic. Double-blind evaluation to control gastrointestinal spasms occurring during radiographic examination. A preliminary report. Western Med. 5:356-358, 1964.

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Brief Summary

INOICATIONS
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For the treatment ot functional bowel/irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis

THESE FUNCTIDNAL OISDRDERS ARE OFTEN RELIEVED BY VARYING COMBINATIONS OF SEDATIVE, REASSURANCE, PHYSICIAN INTEREST, AMELIORATION OF ENVIRONMENTAL FACTORS.

For use in the treatment ot infant colic (syrup)

Final classification of the less-than-effective indications requires further investigation.

CDNTRAINOICATIONS Dbstructive uropathy (for example, bladder neck obstruction due to prostatic hypertrophy); obstructive disease of the gastrointestinal tract (as in achalasia, pyloroduodenal stenosis); paralytic ileus, intestinal atony of the elderly or debilitated patient, unstable cardiovascular status in acute hemorrhage, severe ulcerative colitis; toxic megacolon complicating ulcerative colitis, myasthenia gravis. WARNINGS: In the presence of a high environmental temperature, heat prostration can occur with drug use (fever and heat stroke due to decreased sweating). Diarrhea may be an early symptom of incomplete intestinal obstruction, especially in patients with ileostomy or colostomy. In this instance treatment with this drug would be inappropriate and possibly harmful. Bentyl may produce drowsiness or blurred vision In this event, the patient should be warned not to engage in activities requiring mental alertness such as operating a motor vehicle or other machinery or perform hazardous work while taking this drug. PRECAUTIDNS Although studies have lailed to demonstrate adverse effects ol dicyclomine hydrochloride in glaucoma or in patients with prostatic hypertrophy, it should be prescribed with caution in patients known to have or suspected of having glaucoma or prostatic hypertrophy Use with caution in patients with. Autonomic neuropathy. Hepatic or renal disease. Ulcerative colitis Large doses may suppress intestinal motility to the point of producing a paralytic ileus and the use of this drug may precipitate or aqgravate the serious complication of toxic megacolon. Hyperthyroidism, coronary heart disease, congestive heart failure, cardiac arrhythmias, and hypertension. Hiatal hernia associated with reflux esophagitis since anticholinergic drugs may aggravate this condition

Do not rely on the use of the drug in the presence ol complication of biliary tract disease. Investigate any tachycardia before giving anticholinergic (atropine-like) drugs since they may increase the heart rate. With overdosage, a curare-like action may occur. ADVERSE REACTIONS Anticholinergics/antispasmodics produce certain effects which may be physiologic or toxic depending upon the individual patient's response. The physician must delineate these. Adverse reactions may include xerostomia; urinary hesitancy and retention, blurred vision and tachycardia; palpitations; mydriasis, cycloplegia, increased ocular tension; loss of taste, headache; nervousness; drowsiness; weakness; dizziness; insomnia, nausea, vomiting, impotence; suppression of lactation, constipation, bloated feeling, severe allergic reaction or drug idiosyncrasies including anaphylaxis; urticaria and other dermal manitestations; some degree of mental confusion and/or excitement, especially in elderly persons; and decreased sweating With the injectable torm there may be a temporary sensation of lightheadedness and occasionally local irritation. DOSAGE AND AOMINISTRATION. Dosage must be adjusted to individual patient's needs

Usual Dosage Bentyl 10 mg capsule and syrup: *Adults* 1 or 2 capsules or teaspoonfuls syrup three or four times daily *Children* 1 capsule or teaspoonful syrup three or lour times daily. *Infants* ½ teaspoonful syrup three or lour times daily (May be diluted with equal volume of water) Bentyl 20 mg *Adults.* 1 tablet three or four times daily. Bentyl Injection. *Adults* 2 ml (20 mg.) every four to six hours intramuscularly only NOT FOR INTRAVENOUS USE. MANAGEMENT OF DVEROOSE: The signs and symptoms of overdose are headache, nausea, vomiting, blurred vision, dilated pupils, hot, dry skin, dizziness, dryness of the mouth, difficulty in swallowing, CNS stimulation Treatment should consist of gastric lavage, emetics, and activated charcoal Barbiturates may be used either orally or intramuscularly for sedation but they should not be used if Bentyl with Phenobarbital has been ingested If indicated, parenteral cholinergic agents such as Urecholine® (bethanecol chloride USP) should be used

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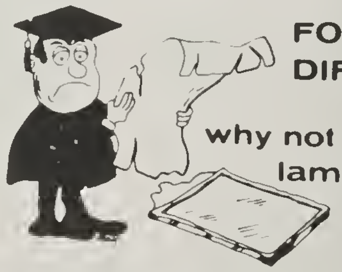

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ArMA Reports

THE MINUTES APPEARING IN THIS SECTION HAVE BEEN EDITED TO CONSERVE SPACE. A COMPLETE COPY OF THE MINUTES OF ANY MEETING WILL BE MAILED TO ANY MEMBER REOUESTING THEM.

EXECUTIVE COMMITTEE

The meeting of the Executive Committee of the Arizona Medical Association, Inc. held at 810 West Bethany Home Road, Phoenix, Arizona on Friday, September 7, 1979, a quorum being present, convened at 6:32 p.m., William E. Crisp, M.D., President and Chairman presiding.

Health Manpower Committee

Dr. Linkner addressed the committee relative to his attendance during the last several months at meeting of the Join Legislative Committee regarding certain legislation which may be proposed in the 1980 session. In connection with those meetings and the request of the Joint Legislative Committee for comments from the several organizations represented, the Health Manpower Committee and formulated and presented to the Executive Committee certain position statements relating to Chiropractic, Naturopathy, Nurse Practitioners and Podiatry for adoption by the Arizona Medical Association and submission to the Joint Legislative Committee at its next scheduled meeting. After a review of the position statements and a lengthy discussion,

It was moved and carried that the position statements relating to Chiropractic, Naturopathy, Nurse Practitioners and Podiatry prepared by the Health Manpower Committee be submitted to counsel for the Association for review and consideration and that the opinion of counsel, together with such statements be presented to the Board of Directors in meeting on October 6, 1979, for its review and final determination.

Arizona Center for Law in the Public Interest and the Gray Panthers Chapter of Sun City

As a point of information only, the committee was informed of a recent request to HEW by the Arizona Chapter for Law in the Public Interest and the Gray Panther Chapter of Sun City for a list of physicians who accepted assignments under Medicare during the first quarter of 1979.

"Survival Guide"

Letter of August 28, 1979 from Mountain Bell relative to the inclusion of a "Survival Guide" in Arizona telephone directories. *Received.*

American Association of Medical Assistants

The committee reviewed the August 13, 1979 letter from the American Association of Medical Assistants and discussed the many items contained therein. It was a consensus of opinion that the New Physician packets presently being utilized by the Association did not lend themselves to the inclusion of any other materials; that the policy of addressing the House of Delegates during the annual meeting of the association was one which was voluntary and any continuation of such policy was at the discretion of AAMA; that the Association would be pleased to consider articles, in parapgraph form, for inclusion in its newsletter, *Medical Memos*; that, while the Association realized the function served by AAMA to its members, it could take no active position relative to membership in such organizaton, nor could it, due to the present commitment of funds, lend financial support for AAMA seminars. It was determined that this consensus be communicated by letter to the American Association of Medical Assistants.

AMA's Speaker to Speaker Meeting

The committee reviewed the August 10, 1979 letter of the AMA regarding a Speaker to Speaker Meeting scheduled to be held December 1, 1979 during the Interim Meeting of the AMA House of Delegates in Honolulu. It was determined that Dr. Price, as the Association's Speaker, would be advised of the place, date and time of such meeting, should he plan to be in Hawaii at that time.

Arizona State Tax Justic Institute

The committee reviewed and discussed the letter of Jerome H. Targovnik, M.D., relative to the formation of an Arizona State Tax Justice Institute to represent the membership in IRS matters. It was determined to advise Dr. Targovnik that counsel for the Association continues to look into the recent interest of the IRS insofar as it relates to the MICA issue.

Voluntary Second Opinion Program

The committee reviewed and discussd the June 26, 1979 request of Metropolitan Life Insurance Company regarding participation in a voluntary second opinion program and Dr. Crisp, as President of the Association, agreed to respond to such letter, setting forth the position of ArMA and the American Medical Association.

Workshop on Dealing and Working with the Media

Dr. Clymer presented to the committee the proposal prepared by the Auxiliary for the holding of a Workshop on Dealing and Working with the Media and, following Discussion,

It was moved and carried to accept the Auxiliary's proposal for co-sponsorship of a workshop on dealing and working with the media to be presented by Mort Enright, Director of AMA Speakers and Leadership Programs from 9 a.m. to 5:00 p.m., Saturday, February 2, 1980, with invitations to be extended, in priority, to state and county officers of the Arizona Medical Association and the Arizona Medical Associaton Auxiliary, with ArMA to bear the expenses incurred in bringing Mr. Enright to the Phoenix area, and with the Auxiliary to finalize all details for such program.

Borrowing Resolution

Mr. Robinson presented to the committee for its approval the Borrowing Resolution required by the Association's lending institution and, following discussion,

It was moved and carried to approve the execution by the Association's officers of the borrowing resolution for a determined line of credit with the Valley National Bank of Arizona.

Professional Guild of Arizona

The committee reviewed and discussed at length the various letters, memos and documents which have been issued and distributed subsequent to the recent industrial Commission of Arizona hearings and the appearance at such hearings by the Professional Guild of Arizona realating to the ICA's fee schedules for Workman's Compensation cases and, additionally discussed what the committee felt to be the existence of a conflict of interest situation with the Chairman of ArMA's Medical Economics Committee, Richard W. Switzer, M.D., being an active member of the Professional Guild of Arizona. Dr. Crisp reported that, in a recent conversation with Dr. Switzer, he had indicated a willingness to resign from such chairmanship.

It was moved and carried that the Association accept the resignation of Richard W. Switzer, M.D. as Chariman of the Medical Economics Committee and that such vacancy be filled at the discretion of the president.

AMA Committee on Continuing Medical Education

The committee received a request for the nomination of Richard L. Collins, M.D., for appointment to the AMA's Committee on Continuing Medical Education.

It was moved and carried that the Association recommend to the

American Medical Association the appointment of Richard L. Collins, M.D. to its committee on Continuing Medical Education.

State of Ariozna Atomic Energy Commission

The committee reviewed a letter from James M. Woolfenden, M.D., Chairman of the State of Arizona Atomic Energy Commission requesting the association's approval of the appointment of Dennis D. Patton, M.D., Michael A. Lawson, M.D., John D. Vosskuhler, M.D., and Gerold A. Kaplan, M.D. to the Commission's Medical Advisory Committee.

It was moved and carried approving the appointment of Dennis D. Patton, M.D., Michael A. Lawson, M.D., John D. Vosskuhler, M.D., and Gerold A. Kaplan, M.D. to the Medical Advisory Committee for the State of Arizona Atomic Energy Commission.

Ad Hoc Membership Committee

Dr. Clymer, Chairman of the Association's Ad Hoc Membership Committee, presented a 1980 Membership Development Plan formulated by the committee in meeting on September 6, 1979. Following a lengthy discussion, it was determined that, while the overall plan seemed to be well-presented and workable one, there were certain areas which could be altered in order to more effectively carry out the program, and

It was moved and carried to approve the 1980 Membership Development Plan submitted by the Ad Hoc Membership Committee, with the exception of the suggested second and third billings and the distribution to nonmembers of Arizona Medicine, Roundup and Sombrero and to direct the Ad Hoc Membership Committee, following deletion of the foregoing items, to put the Association's Development Program into effect.

GOVERNMENTAL SERVICES COMMITTEE

Meeting of the Governmental Services Committee of the Arizona Medical Association, Inc. held September 12, 1979, at 810 W. Bethany Home Road, Phoenix, convened at 7:33 p.m., Louis C. Kossuth, M.D., Chairman, presiding.

Chairman's Report

1979 Amendments to P.L. 93-641

The chairman informed the committee that the resolution introduced by the committee to the Arizona Medical Association House of Delegates on amending P.L. 93-641 to change the provision on indirect providers, was introduced to the American Medical Association House of Delegates as amended and approved. The AMA House of Delegates further amended the resolution and approved it.

Auxiliary HSA Monitoring

Mrs. Clymer and Mrs. Ogden were introduced and welcomed. They informed the Committee that the Auxiliary's program for monitoring HSA meetings is getting off the ground and that they will have more information about it after the meeting of the State Auxiliary's Board to be held at the end of September.

CAHSA Activities

The Chairman spoke briefly about current activities of CAHSA and encouraged all members of the Committee who are not already memebers of their local HSA to join. He also outlined some of the activities of the Department of Health Services and his review of the Statewide Hypertension Program, Statewide Health Education Program and Health Incentive Grants.

The ArMA Symposium For Health Planning

The concept of sponsoring a workshop on health planning was approved by the ArMA Board of Directors. The Committee discussed at length the important elements of the program and developed the following program draft.

Theme: Understanding and Participating in Health Planning.

Date: January 19, 1980.

Location: ArMA Headquarter Offices

Program Format: *Available from ArMA Offices.*

Invitees:

ArMA Board of Directors.
Governmental Services Committee Members.
County Medical Society Presidents, Secretaries, Presidents-Elect, Chairmen of Health Planning Committees, and Staff Members.
Housestaff and Medical Student Representatives.
Officers and Representatives from State and County Medical Society Auxiliaries.
Physician Members of HSA Boards and SHCC.
Spouses.

Pre-conference Packet:

HSA's will be requested to submit information on their HSA, how it works, what committees they have, who sits on those committees, how committees are formed, etc.

It was determined that the Chairman will contact potential speakers and participants and refine the program draft developed.

Health Planning Committee

The Committee was asked to consider, for future discussion, whether ArMA should have a separate Health Planning Committee which could be a section of the Governmental Services Committee. It was recognized that the Governmental Services Committee spends most of its time on health planning and neglects other issues charged to it in the bylaws.

SCIENTIFIC ASSEMBLY COMMITTEE

The meeting of the Scientific Assembly Committee of the Arizona Medical Association, Inc. held at 810 W. Bethany Home Road, Phoenix, Arizona, on Saturday, September 29, 1979, convened at 1:33 p.m., James M. Hurley, M.D., Chairman, presiding.

Annual Meeting Registration Fees

The committee received the action taken by the Board of Directors on August 4, 1979 on the committee's previous recommendation that $10.00 of Association membership dues be allocated and earmarked for annual meeting expenses and that there be no member registration for the scientific portion of ArMA's annual meeting. Due to the fact that the Board action had referred the matter to the Finance Committee for its evaluation and recommendations and that the Finance Committee met early on September 29th, Dr. Hurley requested Dr. Robles, as chairman of the Finance Committee to report his committee's determinations. Dr. Robles advised that following a lengthy discussion and review of past years' annual meeting income and expenses, his committee was recommending that the Board make no changes in the present system used for registration by ArMA members for attending the Arizona Medical Association annual meeting. A lengthy discussion ensued and the committee took the following action:

It was moved and carried that the Scientific Assembly Committee urge the Board of Directors to reconsider having no membership registration fee for the Arizona Medical Association annual meeting, beginning with the meeting to be held in 1981, in order to encourage better member attendance.

Solicitation of Local Papers and Exhibits

The committee was informed that solicitation for local papers and exhibits had been made to all Housestaff individually on 8/31/79; to all Medical Education Directors on 8/31/79; and to all ArMA members on 9/11/79.

Solicitation of Financial Support

The committee was advised of the financial contributions, indications of forthcoming financial support and offers to provide speakers which had, thus far, been received as the result of a solicitation to over 200 pharmaceutical and insurance companies. Following discussion, it was agreed that the committee, after finalization of the program to be provided by the faculty from the University of Colorado Medical School and the choosing of scientific papers, would utilize the offers received from various pharmaceutical companies to provide speakers to fill any slots then remaining uncommitted.

Specialty Society Interest

Arizona Society of Pathologists

The committee reviewed a letter dated 6/28/79 received from the Arizona Society of Pathologists relating to problem areas which might arise by virtue of any future attempt for scheduling specialty society meetings in conjunction with the Association's annual meeting and the committee determined that the matter required in-depth consideration prior to whole committee action thereon. Suresh C. Anand, M.D., James M. Hurley, M.D., and Marvin C. Schneider, M.D. were requested to act as an ad hoc committee to make whatever determinations they deemed feasible with regard to specialty society participation during annual meeting and the questions posed in the 6/28/79 letter and report back to the committee at a future meeting so that formal action might be taken.

Arizona Urological Society

Letter dated 6/7/79 from the Arizona Urological Society. *Received.*

1980 Program Plans

The Pointe Resort

The site plan and layout of the convention center, meeting rooms and grande ballroom of The Pointe Resort, the location of the 1980 Annual Meeting. *Received.*

Program Topics

Topics used for the scientific program of the annual meeting in 1976 through 1979. *Received.*

Program Topics Offered

A. Breakfast Panel on "Confidentiality"

The committee reviewed an offer received from Thomas E. Bittker, M.D. to provide a breakfast panel on "Confidentiality" and, following a lengthy discussion,

It was moved and carried that Thomas E. Bittker, M.D., be invited to attend the next meeting of the Scientific Assembly Committee to outline in greater detail the program on "Confidentiality," prior to any determination by this committee of a time slot allocation in the scientific portion of the 1980 Annual Meeting program.

B. Breakfast Panel on "Litigation Against the Medical Profession"

The committee considered the concept of including a breakfast panel on litigation, which would present much needed information to the physician from both a national and local standpoint, and that committee took the following action:

It was moved and carried that a breakfast panel entitled "Litigation and the Medical Profession" be scheduled for the morning of Saturday, April 26, 1980.

C. Panel on "Consumer & Physician Attitudes Toward Physician Services, Medical Malpractice, and Patients"

The committee reviewed the request of the Ad Hoc Malpractice Crisis Committee relating to the inclusion of a panel on "Consumer & Physician Attitudes Toward Physician Services, Medical Malpractice and Patients" and it was the consensus of opinion that this was a very important issue, requiring greater length of time than allocated to breakfast panels and it was determined that such panel would be included in the program for the 1980 Annual Meeting to be held on Saturday, April 26, 1980, from 3:30 to 5:00 p.m.

D. Panel on "National Hospice Movement"

The Auxilairy informed the committee that they had recently received an offer for a presentation during the annual meeting of a panel on the "National Hospice Movement" but had been advised that the national speaker would desire a longer time period than that allocated for breakfast panels. It was the consensus of opinion that a greater time slot could not be offered during the 1980 program but the Auxiliary was requested to again contact the speaker advising that a breakfast panel was available if he so desired or, alternatively, the panel could be included in a future meeting possibly in conjunction with plenary sessions or interdisciplinary programs.

General Program Schedule

General Program Schedule outlining basic daily format. *Received.*

Detailed Scientific Program Worksheets

Worksheets for development of scientific program. *Received.*

University of Colorado Medical Center Program Proposal

Frank J. Cozzetto, M.D., Associate Dean for Continuing Education at the University of Colorado Medical Center, presented to the committee a list of faculty members and topics for ocnsideration by the committee. A lengthy discussion followed, during which the committe chose to accept the following faculty members: Waston A. Bowes, Jr., M.D., Henry N. Claman, M.D., Michael Johnson, M.D., Alan S. Nies, M.D., Bruce C. Paton, M.D., David S. Pearlman, M.D., Thomas L. Petty, M.D., Ronald Pfister, M.D., William A. Robinson, M.D., Joe M. Sanders, Jr., Lt. Col, M.C. Robert W. Schrier, M.D., Thomas E. Starzl, M.D. and Eng W. Tan, M.D. Additionally, topics selected and time slots filled, pending confirmation by the faculty members.

Auxiliary Program Review

Auxiliary/ArMA Joint Spouse Program

Mrs. Clymer and Mrs. Collins addressed the committee regarding the Thursday morning joint spouse program and indicated that, because of the tremendous response to last year's program, they wished to continue with the theme and entitled the program "Physicians' Families - Three Generations."

It was moved and carried that the Auxiliary/ArMA joint spouse program scheduled for the morning of Thursday, April 24, 2980, be entitled "Physicians' Families - Three Generations"

5,000/10,000 Meter Race

Mrs. Collins and Mrs. Newman informed the committee that plans were progressing nicely for the 5,000/10,000 meter race which will be held early Thursday morning, April 24, 1980 and will be run along the canal at The Pointe.

FINANCE COMMITTEE

The meeting of the Finance Committee of the Arizona Medical Association, held at 810 W. Bethany Home Road, Phoenix, Arizona on Saturday, September 29, 1979, convened at 10:24 a.m., Neopito L. Robles, M.D., Treasurer and Chairman, presiding.

Establishment of 1980 Committee Budgets

The committee reviewed and considered, item by item, the amounts requested by the chairman of all ArMA Committee for the 1980 Committee Department Budget, the total amount of which was established by action of the House of Delegates in the sum of $88,200.00. During a lengthy discussion, the committee also considered the fact that the expenditures of the Underwriting and Calim Review Committees were not really within the purview of the Finance Committee in that these were predetermined contract funds and therefore those committees should be deleted from the budget entirely, and took the following action:

It was moved and carried to recommend to the Board of Directors that the 1980 Committee budgets be as follows:

500A	Articles of Incorporation and Bylaws	$ -0-
500B	Benevolent & Loan	6,000.00
500D	Board of Directors	2,500.00
500E	Executive	2,000.00
500F	Finance	100.00
500H	Grievance	-0-
500I	History & Obituaries	-0-
500J	Maternal & Child Health	1,000.00
500K	Health Manpower	1,800.00
500L	Legislative	15,000.00
500M	Medical Education	4,000.00
500N	Medical Economics	1,200.00
500O	Occupational helath	-0-
500Q	Professional	2,500.00
500R	Public Relations	20,000.00
500S	Governmental Services	1,000.00
500U	House Staff	1,600.00
500W	ArMA Auxiliary	3,000.00
500X	Physicians Rehabilitation	1,000.00
Total 1980 Committee Department Budget		$ 62,700.00

Advisory Council on Nursing Manpower & Education Needs

The committee considered the request from the Advisory Council on Nursing Manpower & Education Needs for funding in the amount of $5,000.00 or more referred to this committee by action of the Board of Directors on August 4, 1979, and subsequent to a lengthy discussion, took the following action.

It was moved and carried that the Arizona Medical Association approve in principle, the study intended to be undertaken by the Advisory Council on Nursing Manpower & Education needs relative to the shortage of nurses within the state, but that no funds are available within the 1979 budget to offer monetary assistance.

Member Registration Fees ArMA Annual Meeting

The committee considered the request of the Scientific Assembly Committee that a portion of ArMA membership dues be allocated and earmarked for annual meeting expenses in order to alleviate a member registration fee for the scientific portion of that meeting. After much discussion and a thorough review by the committee of past years' income and expenses of the annual meeting, it was determined that the only alternative to a member registration fee would be an increase in membership dues and that, at this point in time, such action could not be justified, and the committee took the following action:

It was moved and carried to recommend to the Board of Directors that there be no change in the present system used for registration by ArMA members for attending the Arizona Medical Association Annual Meeting.

Board of Directors

The committee received for its review the action taken by the Board of Directors on February 10, 1979, on recommendations previously made by the Finance Committee.

1981 Budget Planning

Mr. Robinson discussed briefly with the committee the steps previously taken in past years in preparing a draft budget for the committee to rework and finalize, requesting input from the committee as to whether they desire this process be continued or whether some type of change might allow the process to run more smoothly. It was determined that the preparation, analyzation and completion of the 1981 budget would be conducted as it had in the past, whic had proved to be most satisfactory i allowing the committee to perform it function on a timely basis.

Future Medical Meetings

The following institutions and organizations hav been accredited for their continuing medica education programs by the Liaison Committee or Continuing Medical Education

Arizona Heart Institute
Arizona Medical Association
Arizona State Hospital, Phoenix
Camelback Hospital, Phoenix
Desert Samaritan Hospital, Mesa
Good Samaritan Hospital, Phoenix
Health Maintenance Associates, Phoenix
Maricopa County General Hospital, Phoenix
Mesa Lutheran Hospital, Mesa
Phoenix Baptist Hospital & Health Center
Phoenix Indian Medical Center
Phoenix Memorial Hospital
Phoenix Psychiatric Council
St. Joseph's Hospital & Medical Center, Phoenix
St. Lukes's Hospital & Medical Center, Phoenix
St.Mary's Hospital, Tucson
Scottsdale Memorial Hospital
Tucson Hospitals Medical Education Program, Tucson
Veterans Administration Hospital, Phoenix
Veterans Administration Hospital, Prescott
University of Arizona College of Medicine, Tucson

The accredited institutions and organizations above produce a variety of continuing medical education programs. Each accredited institution and organization is responsible for designating which of these programs meet ArMA's requirements for Category 1 credit. Physicians who participate in programs which are designated Category 1 by accredited institutions will receive Category 1 credit toward the ArMA Certificate in CME and the AMA's Physician's Recognition Award.

NOVEMBER

27th Annual Meeting of the States and Mexico.
November 14-17, 1979. Doubletree Inn Tucson, AZ. Sponsor: Medical Society of th United States and Mexico. Contact: Mrs Carolyn Parsons, Executive Secretry, 3161 N Pantano Road, Tucson, AZ 85715.

Arizona PerinatalProgram
November 16-17, 1979. Ramada Inn, Lake Havasu, AZ. Sponsor: Arizona Medical Association. Contact: Arizona Perinatal Program, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 15 hours of category 1 credit.

Advanced Cardiac Life Support (Provider Course)
November 16-17, 1979. Arizona Medical Association, Phoenix, AZ. Sponsor: ArMA & Arizona Affiliate - Amer. Heart Assoc. Contact: Carol Turner, ArMA, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

"Wellness—An Arthritis Reality"
November 17, 1979, Sheraton-Scottsdale Inn. Sponsor: Arthritis Foundation, Central Arizona Chapter. Contact: Diona Siedler, Program Services Coordinator, Arthritis Foundation Central Arizona Chapter, 100 W. Osborn Rd, Ste D, Phoenix, AZ 85013

Arizona Society of Otolaryngology and Maxillo-Facial Surgery
November 16 & 17, 1979. The Wigwam, Litchfield Park, AZ. Sponsor: Arizona Society of Otolaryngology & Maxillo-Facial Surgery. Contact: Neil O. Ward, M.D., 4550 N. 51st. Ave., Phoenix, AZ 85031.

Hypertension
November 17, 1979. Good Samaritan Hospital, Phoenix, AZ. Sponsor: Good Samaritan Hospital. Contact: CME office GSH, 1033 E. McDowell Rd., Phoenix, AZ 85006. Approved for 6 hours of category 1 credit.

American Society of Anesthesiologists (Regional Refresher Course)
November 17-18, 1979. Camelback Inn, Phoenix, AZ. Sponsor: American Society of Anesthesiologists. Call: (312) 825-5586, Frank Connell. Approved for 8 hours of category 1 credit.

The Third Annual National Symposium on Aging
November 17 & 18, 1979. Holiday Inn, Golden Gateway, San Francisco, CA. Sponsor: Univ. of California San Francisco. Contact: Univ. of California, Continuing Education Health Sciences, 24 Kirkham, San Francisco, CA 94143.

Differential Diagnosis of Red Eye
November 28, 1979. Glendale Samaritan Hospital, Glendale, AZ. Sponsor: Good Samaritan Hospital. Contact: GSH, Office of CME, 1033 E. McDowell Rd., Phoenix, AZ 85006. Approved for 1 hour of category 1 credit.

Therapeutic Drug Monitoring
November 28-30, 1979. Del Webb's Townhouse, Phoenix, AZ. Sponsor: American Society of Clinical Pathologists. Contact: 2100 W. Harrison St., Chicago, ILL. 60612. Call (312) 738-1336. Approved for 21 hours of category 1 credit.

Mangement of Thyroid Nodules
November 29, 1979. Williams Air Force Base. Hospital Conf. Rm. Sponsor: USAF Manpower & Personnel Center, Surgeon's Office. Contact: Williams Air Force Base, Chandler, AZ. Approved for 1 hour of category 1 credit.

Sport Medicine Symposium
November 29-30, 1979. Marriott Hotel, Tucson, AZ. Sponsor: U of A College of Medicine. Contact: Office of CME & Outreach, Arizona Health Sciences Center, Tucson, AZ 85724. Approved for 14 hours of category 1 credit.

DECEMBER

22nd Annual Meeting of American Society of Hematology
December 1-4, 1979. Phoenix Civic Center & Hyatt Regency Hotel. Sponsor: American Society of Hematology. Call: Registration Supervisor (609) 848-1000. Approved for 12 hours of category 1 credit.

The Critically Ill Surgical Patient
December 7-8, 1979. Doubletree Inn, Tucson, AZ. Sponsor: Arizona Chapter - American College of Surgeons, U of A College of Medicine. Contact: Ms. Christy Snow. Office of CME & Outreach, Arizona Health Sciences Center, Tucson, AZ 85724. Approved for 11 hours of category 1 credit.

Clinical Ophthalmology
December 7-8, 1979. Arizona Health Sciences Center, Tucson, AZ. Sponsor: U or A College of Medicine. Contact: Office of CME & Outreach, Arizona Health Sciences Center, Tucson, AZ 85724. **ECG Interpretation and Arrhythmia Management**
December 7-9, 1979, Hyatt Regency, Phoenix, AZ. Sponsor: International Medical Education Corp., 64 Inverness Drive East, Englewood, Co 80112. Approved for 15 hours category 1 credit by AMA, 15 prescribed hours by AAFP, and 13 hours category 1 by ACEP.

Occupational medicine for the Practicing Physician - Part I
December 8, 1979. The Adams Hotel, Phoenix, AZ. Sponsor: U of A College of Medicine. Contact: Office of CME & Outreach, Arizona Health Sciences Center. Approved for 7 hours of category 1 CME credit.

8th Annual Seminar: Bone and Joint Diseases
December 8 and 9, 1979, Camelback Inn, Scottsdale, AZ. Sponsor: Maricopa County General Hospital. Contct: Alex Newman, M.D., Program Chairman, Dept. of Radiology, Maricopa County General Hospital, 2601 E. Roosevelt Street, Phoenix, AZ 85008. Approved for 14 hours of category 1 credit.

Aphasia
December 12, 1979. Maryvale Samaritan Hosptial. Sponsor: Good Samaritan Hospital. Contact: CME office GSH, 1033 E. McDowell Rd., Phoenix, AZ 85006 Approved for 1 hour of category 1 credit.

Approaching the Emergent Patient
December 13, 1979. Williams Air Force Base. Hospital Conf. Rm. Sponsor: USAF Manpower Personnel Centert, Surgeon's Office. Contact: Williams Air Force Base, Chandler, AZ. Approved for 2 hours of category 1 credit.

ACLS Provider Course
December 14 & 15, 1979. ArMA Offies, 810 W. Bethany Home Rd., Phoenix, AZ. Sponsor: Arizona Affiliate of the American Heart Assoc. Contact: Carol Turner, ArMA Offices 810 W. Bethany Home Road, Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

1980

JANUARY

Impotence
January 9, 1980. Maryvale Samaritan Hospital. Sponsor: Good Samaritan Hospital. Contact: CME office, Good Samaritan Hospital, 1033 E. McDowell Road, Phoenix, AZ 85006. Approved for 1 hour of category 1 credit.

Evaluation & Treatment of Coagulation Disorders
January 10, 1980. Williams Air Force Base, Hospital Conf. Rm. Sponsor: USAF Manpower & Personnel, Surgeon's Office. Contact: Williams Air Force Base, Chandler, AZ. Approved for 1 hour of category 1 credit.

OBGYN Seminar
January 10-11, 1980. Arizona Health Sciences Center, Tucson, AZ. Sponsor: U of A College of Medicine. Contact: Office of CME and Outreach, Arizona Health Sciences Center, Tucson, AZ 85724

General Pediatrics #1
January 10-12, 1980. Doubletree Inn, Scottsdale, AZ. Sponsor: American Academy of Pediatrics. Contact: Ms. Judy C. Hambourger, Dept. of Education, American Academy of Pediatrics. P.O. Box 1034, Evanston, Ill 60204. Approved for 18 hours of category 1 credit.

Diagnosis & Treatment of Headache
January 11-13, 1980. La Posada, Scottsdale, AZ. Sponsor: American Association for Study of Headache. Call: Robert Kunkel, M.D. (216) 444-5654. Approved for 15½ hours of category 1 credit.

Case Studies In Emergency Care
January 12, 1980. The Adams Hotel, Pheonix, AZ. Sponsor: U of A College of Medicine. Contact: Office of CME and Outreach, Arizona Health Sciences Center, Tucson, AZ 85724. Approved for 7 hours of category 1 credit.

Advanced Cardiac Life Support Program (Provider Course)
January 11-12, 1980. Arizona Medical Association, Phoenix, AZ. Sponsor: ArMA & Arizona Affiliate - American Heart Assoc. Contact: Carol Tuner, ArMA Offices 810 W. Bethany Home Road, Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

23rd. Annual Cardiac Symposium
January 18-19, 1980. Mountain Shadows Resort, Scottsdale, AZ. Sponsor American Heart Association & Maricopa County General Hospital. Call: Charlotte Beeler 277-4846.

8th Annual Surgical Symposium
January 21-25, 1980. Doubltree Inn, Scottsdale, AZ. Sponsor: Maricopa County General Hospital, & Phoenix Surgical Society. Call: Susie Davis 264-9021. Approved for 31 hours of category 1 CME credit.

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PRECAUTIONS: Thrombocytopenic purpura may follow the administration of quinine in highly sensitive patients. Recovery will follow withdrawal of the medication. Cinchona alkaloids, including quinine, have the potential to depress the hepatic enzyme system that synthesizes the vitamin K-dependent factors. The resulting hypoprothrombinemic effect may enhance the action of warfarin and other oral anticoagulants.

ADVERSE REACTIONS: Aminophylline may produce intestinal cramps in some instances, and quinine may produce symptoms of cinchonism, such as tinnitus, dizziness, and gastrointestinal disturbance. If ringing in the ears, deafness, skin rash, or visual disturbances occur, the drug should be discontinued.

DOSAGE AND ADMINISTRATION:
1 tablet upon retiring. When necessary, 1 additional tablet may be taken following the evening meal.

Product Information as of September, 1977

U.S. Patent 2,985,558

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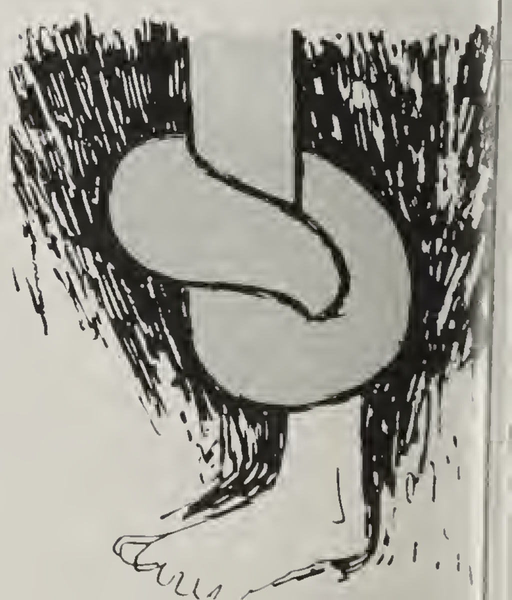

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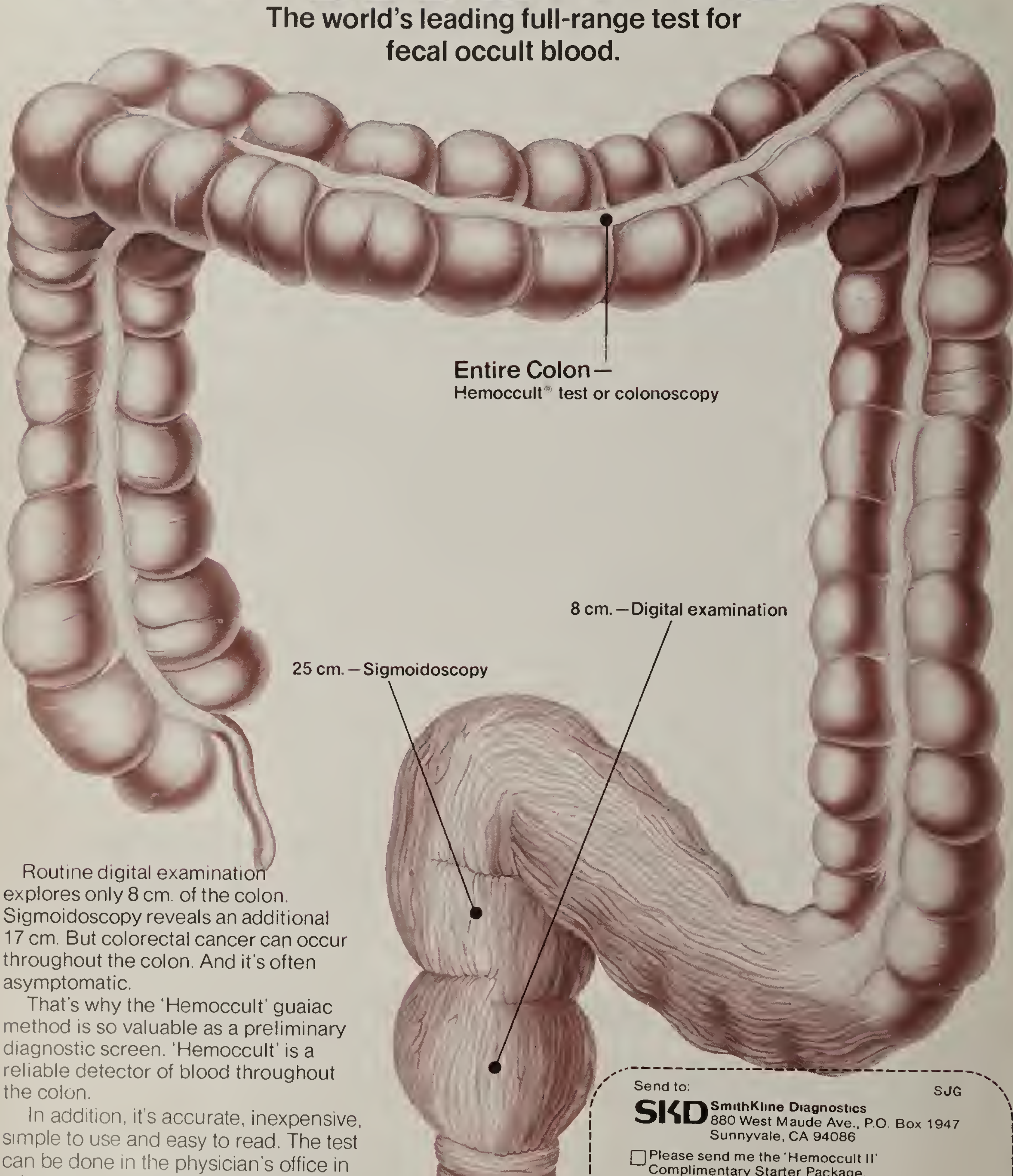

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Recognition of Problems of the Hand
January 23, 1980. Glendale Samaritan Hospital, Glendale, AZ. Sponsor: Good Samaritan Hospital. Contact: Good Samaritan Hospital Office of CME, 1033 E. McDowell Rd., Phoenix, AZ 85006.

Medical Aspects of Family Planning
January 24, 1980. Williams Air Force Base, Hospital Conf. Rm. Sponsor: USAF Manpower & Personnel Center & Surgeon's Office. Contact: Williams Air Force Base, Chandler, AZ. Approved for 1 hour of category 1 credit.

Sleep Disorders
January 26, 1980. Good Samaritan Hospital, Phoenix, AZ Sponsor: Good Samaritan Hospital. Contact: GSH, CME office, 1033 E. McDowell Road, Phoenix.AZ 85006. Approved for 6 hours of category 1 credit.

27th Annual Scientific Assembly of AAFP
January 20-Feb.2, 1980. Doubletree Inn, Scottsdale, AZ. Sponsor: U of A College of Medicine, & AAFP. Call: Lee Mahoney 949-7797. Approved for 19 hours of category 1 credit.

FEBRUARY

New Perspectivies in Cerebral Localization
February 7-9, 1980. Hyatt-Regency Hotel, Phoenix, AZ. Sponsor: St. Joseph's Hospital and Medical Center, Barrows Neurological Institute. Contct: Richard A. Thompson, M.D., Chairman, 350 West Thomas Rod, Phoenix, AZ 85013.

Coronary Disease, Exercise Testing & Cardiac Rehabilitation
February 8-10, 1980. Mountain Shadows Resort, Scottsdale, AZ. Contact International Medical Education Corp., 64 Inverness Drive East, Englewood, Co 80112. Approved for 13 hours category 1 credit by AMA, and 13 prescribed hours by AAFP

5th Annual Seminar - Radiology & Geriatric Medicine
February 8-10, 1980. Doubletree Inn, Scottsdale, AZ. Sponsor: Maricopa County General Hospital. Contact: Austin R. Sandrock, M.D., Program Chairman, Maricopa County General Hospital, Dept. of Radiology, 2601 East Roosevelt Street, Phoenix, Z 85008. Approved for 15 required hours of category 1 credit.

Coronary Disease, Exercise Testing & Cardiac Rehabilitation
February 8-10, 1980. Moutain Shadows Resort, Scottsdale, AZ. Contact: International Medical Educ. Corp., 4 Inverness Dr. East, Englewood, CO 80112. Approved for 13 hours of prescribed CME credit by AAFP.

Taos Medical Symposium
February 12-15, 1980. Tennis Ranch of Taos, Taos, New Mexico. Sponsor: New Mexico Chapter, AAFP, CME Committee. New Mexico Medical Society, Albuquerque Academy of Family Physicians, Albuquerque & Bernalillo County Medical Association. Contact: Thomas A. Bodnar, Program Director, 2650 Yale Blvd., S.E., No. 103, Albuquerque, New Mexico 87106

Nineth Annual Day of Medicine: Day of Cardiology
February 15, 1980. Arizona Biltmore, Phoenix, AZ. Sponsor. St. Joseph s Hospital & Medical Center, & Flinn Foundation. Contact Darlene Graham, Dept. of IM, St. Joseph's Hospital, P.O. Box 2071, Phoenix, AZ 85001 Approved for 6 hours of category 1 credit.

2nd Annual Ski & Study Program
February 16-21, 1980. Park City, Utah. Sponsor: Desert Samaritan Hospital CME Committee. Contact: Betty Wood Desert Samaritan Hospital, Mesa, AZ.

Eighth Annual Taos Lung Disease Symposium
February 22-24, 1980. Sponsor: New Mexico Thoracic Society. Contact: New Mexico Thoracic Society, 216 Truman N. E., Albuquerque, N. 87108. Approved for 13½ hours of category 1 credity by AMA.

Second Annual International Cataract Surgery Symposium
February 18-20, 1980. Sponsor: Arizona Foundation for Ophthalmic Research and Education. Contact: Ronald W. Barnet, M.D., Medical Director, 421 N. 18th St., Ste 105, Phoenix, AZ 85006. (602) 257-1077. Approved for 12 to 22 hours of category 1 credit.

8th Annual Medicine Conference Advances in Internal Medicine II
February 21-23, 1980. Carefree Inn & Resort, Carefree Arizona 85008. Sponsor: Maricopa County General Hospital. Contact: Myra DaBolt, 2601 E. Roosevelt St., Phoenix, AZ 85008. Approved for 21 hours category 1 credit.

Gastroenterology for Clinicians - New Directions
February 21-23, 1980. Del Webb's TowneHouse, Phoenix, AZ. Sponsor: David C.H. Sun Memorial Institute. Contact: Mrs. David C.H Sun, David C.H. Sun Memorial Institute, 4129 East Sandy Mt. Rd., Scottsdale, AZ 85253.

Pediatric Update 1980
February 25-28, 1980. Sponsor: St. Joesph's Hospital and Medical Center, Dept. of Pediatrics. Contact: Ronald A. Christensen, M.D., Dept. of Pediatric Education. St. Joseph's Hospital and Medical Center, 350 W. Thomas Rd., Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

Third Annual Mid-Winter Symposium in OB/GYN
February 27- 29, 1980. Scottsdale Hilton Hotel, Scottsdale, AZ. Sponsor: Maricopa County Hospital, Dept. of OB/GYN and Phoenix OB/GYN Society. Contact: The Secretary, Phoenix, OB/GYN Society, 333 East Virginia, Ste 222, Phoenix, AZ 85004.

New Mexico Metabolism Symposium
February 28-March 1, 1980. Taos, New Mexico. Contact: Office of Education. Lovelace Medical Center, Albuquerque, New Mexico. (505) 842-7353.

Contemporary Managment of Stroke and Coronary Disese
February 27 - March 1, 1980. Lodges in Taos, New Mexico. Sponosr: Symposia de Santa Fe. Contact. W.J. Levy, M.D., Symposia de Santa Fe, P.O. Box 5175, Santa Fe, New Mexico 85702

MARCH

2nd Annual Symposium on Current Concepts in the Managment of Acute and Chronic Pain
March 1-2, 1980. Hyatt Regency, Phoenix, AZ Sponsor: Maricopa County General Hospital. Contact: George Wallace, M.D., Program Director, MCGH, 2601 E. Roosevelt, Phoenix, AZ 85008.

Durango, Colorado Ski Symposium
March 5 and March 9, 1980. Sponsor: Albuquerque Academy of Family Physicians. Conact: Albuquerque Academy of Family Physicians, c/o New Mexico Medical Society, 2650 Yale Blvd., S.E., Ste 105, Albuquerque, New Mexico 87106.

Sixteenth Annual Arizona Chest Symposium
March 28-30, 1980. Doubletree Inn, Tucson, Arizona. Sponsor: University of Arizona College of Medicine, and Tucson Medical Center. Contact: Linda Alpert, R.N., Symposium Coordinator, Tucson Medical Center, P.O. Box 6067, Tucson, AZ 85733. Approved for category 1 credit.

APRIL

Emergency Room Radiology-Revised

April 11-13, 1980. Arizona Biltmore, Phoenix, Arizona. Sponsor: Maricopa County General Hospital. Contact: Austin R. Sandrock, M.D., Program Chairman, Maricopa County General Hospital, 2601 E. Roosevelt St., Phoenix, AZ 85008

MONTHLY OR WEEKLY

Shrine Medics Meeting
Second Tuesday of each month, Doctors Hospital, 5:45 p.m. J. South Classroom. Sponsor: Shrine Medics. Contact: Robert C. Briggs, M.D., 5121 N. Central Ave., Phoenix, AZ 85012.

Office Psychiatry for the Primary Provider
2nd Monday of month, 4811 N. 7th Street, Phoenix, AZ. Sponsor: Arizona Health Plan. Contact: T.R. Bittker, M.D., Box 5000, Phoenix, AZ 85010. Approved for category 1 credit.

Counter Transference Group
Weekly, Thurs. 8—10 p.m. Sponsor: Phoenix Psychiatric Council. Contact: James E. Campbell, M.D., 5051 N. 34th St., Phoenix, AZ 85018. Approved for 2 hours category 1 credit.

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Monthly Meeting of Tucson Radiologists
Last Tues. of month. Plaza International, Tucson, AZ. Sponsor: U of A Medical Center, Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., U of A Health Sciences Center, Dept. of, Radiology, Tucson, AZ 85724. Approved for 2 hours category 1 credit.

Pediatric Grand Rounds
Tuesday 7:30-8:30 a.m in Phoenix: 1st Tues.—Phoenix Indian Hospital, 2nd Tues.—Maricopa County Hospital, 3rd Tues.—Good Samaritan Hospital, 4th Tues.—St. Joseph's Hospital. Sponsor: Maricopa County Hospital (Phoenix Hospital's Affiliated Pediatric Program). Contact: J. Kipp Charlton, M.D., 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 1 hour per session category 1 credit.

Review of Forensic Pathology
Current Case, Special Topics
Thursday, weekly, 11 a.m., 120 S. 6th Ave., Phoenix, AZ. Sponsor: Arizona Society of Pathologists. Contact: H.H. Karnitschnig, M.D., 120 S. 6th Ave., Phoenix, AZ. Approved for 1 hour per session category 1 credit.

ARIZONA HEART INSTITUTE

4800 N. 22nd St., Phoenix, P.O. Box 10,000, Phoenix, AZ 85064. Contact: Edward B. Diethrich, M.D., Medical Dir.

Clinical Conference Cardiovascular Medicine
Third Tues., second floor classroom

ARIZONA STATE HOSPITAL

2500 E. Van Buren, Phoenix, AZ. 85008. Contact: Arnold L. Kendall, M.D.

A.S.H. Psychiatric Grand Rounds
2nd Wed., 1:00-2:00 P.M., J-6 Conf. Rm., Contact: Dr. Conger & Staff
Clinical-Pathological Conference
3rd Wed., 1:30-2:30 P.M. General Services Bldg., Conf. Rm.
Medical Grand Rounds
4th Wed., 1:00-2:00 P.M., Medical Bldg. Conf. Rm.

BARROW NEUROLOGICAL INSTITUTE

Medical Education
Barrow Neurological Institute of St. Joseph's Hospital and Medical Center, 350 W. Thomas Rd., Phoenix, AZ 85013. Sponsor: St Joseph's Hospital & Medical Center. Contact: John R. Green, M.D. Approved for 1 hour category 1 credit.

Neurology Teaching Conference
Tuesdays, 8:30-9:30 a.m., Eighth Floor Conf. Room.

Neurosurgery Morbidity Conference
Wednesdays, 8:15-9:15 a.m., on first and third weeks, Eighth Floor Conference Room.

Neuro-ophthalmology Conference
Tuesdays, 7:30 a.m. in 8th floor neurology conference room.

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Spinal Injury Conference
Wednesdays, 8:15-9:15 a.m., on second and fourth weeks, in Neuropathology Conf. Rm.—a multidisciplinary review of admission by neurosurgeons, orthopedists, and rehabilitation specialists

Neuro-opthalmology Conference.
Weekly, 7:30 a.m., 8th floor conference Rm.

Neuropathology of Gross Specimens Conference
Thursday, 7:30-8:30 a.m. in the Morgue.

Neurology-Neurosurgical
Fridays, 8-9 a.m., First Floor Conference Rm.

Neuropathology or Neuroradiology Conferences
Friday, 9 a.m., Neuropathology in Neuropathology Conference Rm., Neuroradiology in First Floor Conference Rm.

Neurorehabilitation Conference
Tuesdays, noon, 8th Floor Conference Rm.

Neurosurgical Journal Club
Saturdays, 9-11 a.m. in Eighth Floor Conference Rm.

CAMELBACK HOSPITAL

5055 N. 34th St., Phoenix, AZ 85018. Sponsor: Camelback Hospital. Contact: Medical Director.

Clinical Conference
3rd Tuesday, 8-9 a.m.

DESERT SAMARITAN HOSPITAL

1400 South Dobson Road, Mesa, Arizona, Contact: L.A. Rosati, M.D. Approved for category 1 credit.

Clinical Conference - Dept. of Medicine
Weekly Thurs., 12:30-1:30 P.M. Buffet Lunch, Grande Rm.
Medical Education Programs
Wed.,7:00 P.M. Desert Samaritan Hospital, Mesa.

OB/GYN Medical Staff Conference
Weekly, Monday, 12:30-1:30 P.M. Grande Rm.

GOOD SAMARITAN HOSPITAL

1033 East McDowell Rd., Phoenix, AZ Contact: Warren A. Hinchcliffe, M.D. Approved for category 1 credit.

Anatomical Path. & Cytology
Weekly, Mondays 4:00-5:00 P.M., S-4 Conf Rm.
Gynecological Section Conference
2nd Monday, 12:30-1:30 P.M., West Rm
Monday Noon Conference
Weekly, 12:00-1:00 P.M. R-2 Conf. Rm.
Mortality/Morbidity, Surgery Comm.
2nd Monday, 11:30-2:00 P.M., West Rm.
Obstetrical Section Conference
1st Monday, 12:30-1:30 P.M., West Rm.
Pulmonary Disease Grand Rounds
Weekly, 12:00-1:00 P.M., D-5 North
Family Practice Conference
Weekly Tuesday, 12:00-1:00 P.M., Fam. Practice Conf. Rm.

Pediatric Grand Rounds
3rd. Tuesday, 7:300-8:30 A.M., West Rm.
Surgical Grand Rounds
Weekly. Tuesday 7:00-8:30 A.M. East Rm.
Clinical Cancer Fourm
3rd Wednesday, 12:00-1:00 P.M., West Rm, Contact: John A. Bruner, M.D.
Combined Cardiovascular & Family Practice Conf.
Ist, 2nd, & 4th Wed., 12:00-1:00 P.M., D-5 North.
Dr. Finger's Lecture
Weekly, Wed., 12:30-1:30 P.M., R-2 Conf. Rm.
Learning Disabilities
Weekly, 10:30-12:30 A.M., R-2 Conf. Rm.
OBGYN Dept. Review & Statistics
Weekly, 12:30-3:00 P.M., OBGYN Conf. Rm.
PM & R Grand Rounds
1st. & 3rd. Wed., 12:00-1:00 P.M., R-2 Conf. Rm.
Gynecological Pathology
Weekly Thursday, 1:00-2:00 P.M., S-4 Conf. Rm.
GI Radiology Conference
1st Thursday, 12:00-1:00 P.M., F151, Radiology Conf. Rm.
Phoenix Ultrasound Soc. Meeting.
Medical Education Building, Contact: Dr. Ben Ora
Neurology Grand Rounds
Weekly Thursday, 9:00-10:00 A.M., D-5 North.
Tumor Treatment Planning Conf.
2nd & 4th Thursdays, 12:00-1:00 P.M., F151 Radiology
Joint Tumor Gyn Conference
2nd Friday, 12:30-1:30 P.M., West Rm.
Kinesology
Weekly Friday, 4:00-5:00 P.M., R-2 Conf Rm.
Medical Grand Rounds
Weekly Friday, 8:00-9:00 A.M., West Rm.
Neuropathy Conference
Last Friday, 1:15-2:15 P.M., S-4 Morgue.
Psychiatry Grand Rounds
Weekly Friday, 11:30-1:00 P.M. , East Rm.
PM & R Journal Club
Alt. Fridays, 12:00-1:00 P.M., R-2 Conf. Rm.

KINO COMMUNITY HOSPITAL

2800 E. Ajo Way, Tucson, AZ 85713. Contact: Eric G. Ramsay, M.D. Approved for category 1 credit.

Attending's Hour
5th Monday, 12:30 P.M., Contact: Joseph Goldberger, M.D.
Infectious Disease Conference
3rd Monday, 12:30 P.M., Contact: Larry Lincoln, M.D.
Nephrology Conference
1st Monday, 12:30 P.M., Contact: Alan Cohn, M.D.
OB/GYN Dept. Meeting
Weekly Mondays, 7:30 A.M., Contact: Lewis Shenker, M.D.
Psychiatric Conference
2nd & 4th Mondays, Contact: Jose Santiago, M.D.
Surgical Conference
Weekly Mondays, Contact: Ronald Fischer, M.D.
Cardiology Conference
4th Tuesday, 12:30 P.M., Contact: Brendan Phibbs, M.D.
Gynecology Conference
5th Tuesday, 12:30 P.M., Contact Lewis Shenker, M.D.
Neurology Conference
1st & 3rd. Tuesday, 12:30 P.M., Contact: H.W. Buschsbaum, M.D.
Radiology Conference
2nd Tuesday, 12:30 P.M., Contact: Marvin Baker, M.D.
Clinical Pharmacology
4th Wed., 12:30 P.M., Contact: Lee Gardner, Pharm. D.
Clinical Pathology Conf.
2nd Wed., 12:30 P.M., Contact: Philip Altman, M.D.
Gynecology Pathology Conference
Weekly Wed., 7:45 A.M., Contact: Lewis Shenker, M.D.
Medical Conference-Mortality & Morbidity
3rd Wed., 12:30 P.M., Contact: Joseph Goldberger, M.D.
Rheumatology Conference
1st Wed. 12;30 P.M., Contact: George Werner, M.D.
Cardiology Conference
1st Thursday, 12:30 P.M., Contact: Brendan Phibbs, M.D.
Clinical Correlation
3rd Thursday, 12:30 P.M., Contact: Elliot Munjack, M.D.
G-I Conference
4th Thursday, Contact: Ray Moldow, M.D.
OB/GYN Sonography/X-ray Conference
Weekly Thursday, 9:15 A.M., Contact: Lewis Shenker, M.D.
Chest Conference
2nd & 4th Fridays, 12:30 P.M., Contact: David Engelsberg, M.D.
Dermatology Conference
3rd Friday, 12:30 P.M., Contact: Peter Lynch, M.D.
Hematology Conference
1st Friday, 12:30 P.M., Contact: Steven Ketchel, M.D.

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Every other Wednesday, 7-8 A.M.

MARICOPA COUNTY GENERAL HOSPITAL

2601 E. Roosevelt, Phoenix, Az 85008. Contact: Steven Schaffner, M.D.

Medicine Conference
Daily 12:00-1:00 P.M., Contact: Steven Schaffner, M.D.
Chest Surgery Conference
Weekly Mondays, 1:30 P.M., Satan Cruz Rm.
Obstetrical Problem Conference
Weekly Tuesday, 7:15 A.M., Yavapai Rm
Orthopedic Conference
Weekly Tuesday, 7:30 A.M., Santa Cruz Rm.
Pediatric Grand Rounds
2nd Tuesday, 7:30-8:30 A.M.
Urology Discharge Planning Conference
Weekly,11:30 A.M., Station 42
Hand Surgery Conference
Weekly Wed., A.M., Santa Cruz Rm.
Neurosurgery Discharge Planning
Weekly Wed., 1:30 P.M., Station 42.
OB/Neonatology Seminar
Weekly Wed. 7:15 A.M., Yavapai Room
Clinical Psychiatric Conference
Weekly, Wed., 11:00-12:00 P.M., Menta Health Annex, Rm 1346
Surgery Conference
Weekly Wed., 7:00-8:00 A.M., Surgical Dep
Cardiology Conference
Weekly Thurs. 2:00 P.M., Santa Cruz Room
OB/GYN Resident Conference
Weekly Thurs. 12:00 P.M., Yavapai Rm.

GYN, Endocrine Seminar
Weekly Friday, 12:30 P.M., Santa Cruz Rm.
GYN Grand Rounds
Weekly Friday, 1:30 P.M., Santa Cruz Rm.
OB/GYN Surgical Pathology Conf.
Weekly Friday, 7:00 A.M., Yavapai Rm.
Orthopedic X-Ray Conference
Weekly Friday, 7:30 A.M., Santa Cruz Rm.

PHOENIX BAPTIST HOSPITAL & MED. CENTER

6025 N. 20th Ave., Phoenix, AZ 85015 Contact: James L. Grobe, M.D. Approved fo category 1 credit.

Medical & Surgical Conference
Weekly Monday, 12:00 Noon
Medical Conference
1st. & 3rd Tuesdays, 12:00 Noon.
Medical & Surgical Conference
Weekly Wed., 12:00 Noon.
Medical & Surgical Conference
Weekly Friday, 12:00 Noon.
OB/GYN Section Conference
3rd & 4th Mondays, 1st Floor Conference Room

PHOENIX INDIAN MEDICAL CENTER

4212 North 16th St. Phoenix, AZ 85016 Contact: Leland L. Fairbanks, M.D. Approved for category I credit.

Pediatric Grand Rounds
1st Tuesday, 7:30-8:30 A.M., Pediatric Department
Clinical Staff Teaching Conference
Weekly Wed., 8:00-9:00 A.M., Conf. Rm. A.

PHOENIX MEMORIAL HOSPITAL

1201 S. 7th Ave. Phoenix, AZ. 85036. Contact: George Scharf, M.D., Approved for Category 1 credit.

Monthly Medical Education Seminar
3rd Monday, 6:30 P.M., Kiva Conf. Rm.
Tumor Board Conference
Weekly Friday, 12:00 P.M., Kiva Rm., Contact: Howard Kimball, M.D.

SCOTTSDALE MEMORIAL HOSPITAL

7300 East 4th St., Scottsdale, AZ 85251. Contact: W. S. Williams, M.D. Approved for category 1 credit.

Family Practice Conference
1st Monday, 12:30 P.M., Doctor's Lounge
Morbidity & Mortality Conference
2nd Monday, 12:30 P.M., Doctors' Lounge
Neurology & Neurosurgery Conference
3rd Monday, 12:30 P.M., Doctors' Lounge
Internal Medicine-Clinical Pathlogical Conference
4th Monday, 12:30 P.M., Doctors' Lounge
Pulmonary Medicine Conference
1st Tuesday, 12:30 P.M., Doctors' Lounge
Cardiology Conference
2nd Tuesday 12:30 P.M., Doctors' Lounge
Surgery Departmental Meeting
3rd Tuesday, 12:15 P.M., Doctors' Lounge
Thoracic & Vascular Surgery Conference
4th Tuesday, 12:30 P.M., Doctors' Lounge
Cardiopulmonary Conference
4th Tuesday, 12:30 P.M., Doctors' Lounge
OB/GYN Depart. Meeting & Conference
2nd Wed., 12:30 P.M., Doctors' Lounge
Trauma Conference
3rd Thurs., 12:30 P.M., Doctors' Lounge
Tumor Conference
4th Thursday, 12:30 P.M., Doctors' Lounge
Psychiatry & Behavioral Sciences Conference
1st Friday, 12:30 P.M., Doctors' Lounge
GI Medical-Surgical-Radiological Conference
2nd Friday, 12:30 P.M., Doctors' Lounge
Uronephrology-Oncology
Last Friday, 12:30 P.M., Doctors' Lounge

ST. JOSEPH'S HOSPITAL PHOENIX

350 West Thomas Road, Phoenix, AZ 85013. Contact: Joseph C. White, M.D.

OB/GYN Section Conference
3rd & 4th Mondays, 12:30-1:30 P.M., 1st Floor Conf. Rm.
Medical Grand Rounds
Weekly, Wed., 8:00 A.M., 1st Floor Conference Rm.
Pediatric Grand Rounds
4th Tuesday, 7:30-8:30 A.M., Contact: J. Kipp Charlton, M.D.

ST MARY'S HOSPITAL & HEALTH CENTER

1601 W. St. Mary's Road, Tucson, AZ 85703. Contact: see below.

Monthly Specialty Conference-Dept. of Surgery
1st Monday, 7:30 A.M., Century Rm. A. Contact: Med. Staff Office.
Grand Rounds: Medical Surgical, Family Practice, Pathology, Radiology
Weekly Thursday
Cardiology Conference
Weekly Friday, 8:00-9:00 A.M., Century Rm. Contact: Anthony Forte, M.D.

VETERANS ADMINISTRATION HOSPITAL

7th St. and Indian School Rd., Phoenix, AZ 85012. Sponsor: VA Hospital. Contact: Alfred Heilbrumn, M.D. Approved for Category 1 credit.

Cancer Symposium
Weekly Wed., 1:00 P.M., Contact Dr. Berkley, Ext. 419
Cardiology Conference
2nd Thursday — 1 p.m. — T5, Dr. Habibzadeh, Ext. 348.

Dermatology Conference
1st, 2nd, & 3rd Wednesday 10 A.M. — C118, Dr. Roberts, Ext. 426.

G.I. Clinic
2nd & 4th Wed, 1:00-4:00 P.M., GI Ward
G.I. Radiology Clinical Correlation Conference
1st & 3rd Monday,1:00 P.M., D435.
G.I. Grand Rounds
Weekly, Wed., 8:00 A.M., G-I Ward , Contact: Dr. Severino
G.I. Resident Conference
Weekly Friday, 1:00 P.M., D446, Contacts: Drs. Sanowski & Kozarek
G.I. Rounds
Weekly Thursday, 1:00 P.M., D446
Medical Service Grand Rounds
Weekly Friday — 11 a.m. — T5, Dr. Zeller, Ext 272

Medical-Surgical G.I. Conference
1st & 3rd Monday, 3:00 P.M., Room 3134
Medical-Surgical G.I. Conference
1st & 3rd Friday, 2:00 P.M., Rm. 2128, Contact: Drs. Kozarek and Mertz, Ext 493
Medical-Surgical Chest Conference
1st and 3rd Thursday — 1 p.m. — T5, Dr. Chowdhury, Ext. 388.
Surgical X-Ray Conference & Bedside Teaching Rounds
Weekly, Monday, Room 3134.
Surgery-Pathology Conference
Weekly Thursday, 2 p.m. — 2128, Drs. Mertz, Ext. 493 & Likos, Ext 623

TUCSON VETERANS ADMINISTRATION HOSPITAL

3601 South Sixty Ave., Tucson, AZ 85723. Contacts listed below. Approved for category 1 credit.

Medical Surgical Chest Conference
Weekly Tuesday, 2:00 P.M., Contact: Dr. Young
Medical Grand Rounds
Weekly Wed., 12:00-1:00 P.M., V.A. Hospital Staff Conf. Rm. & (AHSC), Contact: Jay Smith, M.D.
Surgical Grand Rounds
Weekly Wed., 4:00 P.M., Contact: Dr. Putnam
Endocrinology Seminar
1st.,3rd, & 5th Thurdsays, 12:00-1:00 P.M., Rm. N318, Contact: Dept. of Internal Medicine.
Vascular Radiology, Interesting Case Conf.
Weekly Thursday, 12:00 Noon
Neurology Grand Rounds
Weekly, Friday, 12:00 P.M., Contct: Dr. Sibley.

U OF A HEALTH SCIENCES CENTER

Sponsor: U of A College of Medicine, Tucson, AZ 85724. Contact: See below. Approved for category 1 credit.

Anesthesiology Board Review Conference
2nd & 4th Monday, 4:00-5:00 P.M., AHSC Dining Rm. C&D, Contact: Dr. Vaughn & Kryc.
Anesthesiology Basic/Clinical Sciences Lectures
Weekly Thursday, 4:00-5:00 P.M., Room 5403
Anesthesiology Case Discussion
Weekly Wed., 7:00 A.M., AHSC, Dining Rm. C&D.
Anesthesiology Resident Presentation
1st Monday, 4:00-5:00 P.M., AHSC Dining Room, C&D, Contact Drs. Otto & Zehngut.
Cardiac Catheterization Conference
Weekly Friday, 4:00 P.M., Contact: Dr. Temkin.
Cardiology Conference
Weekly Monday, 12:00 P.M., AHSC, Contact: Dr. J. Fernandez
Cardiology Research Conference
Weekly Tuesday, 7:30 A.M., Contact: Dr. Roeske.
Tucson Cardiovascular Society
1st Thursday, 6:00 P.M., AHSC, Contact: Dr. Byrne-Quinn
Cerebrovascular Disease Conference
Weekly Monday, 5:00-6:00 P.M., AHSC, Rm. 5505, Contact: Dr. W. Moore
Clinical Immunology, Allergy & Rheumatology Rounds
Every Friday, Noon-1 P.M. Contact: John Boyer, M.D., Dept. of Internal Medicine.
Cerebrovascular Disease Conference
Mondays, 5-6 P.M., weekly, Rm. 5505. Contact: Wesly Moore, M.D.,Depts. of Surgery, Nurology & Radiology.
Chest Conference
3rd Thursday, 12:00 Noon, TMC, Contact: Dr. Nevin or Dienter
Dermatology Conference
4th Monday, 5:15 P.M., AHSC, Contact: Dr. R. Friedman.
Dermatology Conference
1st Thursday, 12:00 Noon, TMC, Contact: Dr. R. Friedman.
Dermatology Rounds
Weekly Wed., 11:30 A.M., Contact: Dr. Lynch
Endocrinology Seminar
Weekly Thursday, 12:00-1:00 P.M., Contact: Dr. Johnson
Family Practice Department Meeting
1st. & 3rd Wed., 12:30 P.M., TMC, Contact: Dr. O. Koenig
GI Conference
Weekly Friday, 12:00 P.M., TMC, Contact: Dr. C. Krone
GI Pathology conference
4th Friday, 1:30 P.M., AHSC, Contact: S. Paplanus
GI Radiology Conference
Weekly Monday, 7:30 A.M., AHSC, Contact: Dr. T. Hunter
Head & Neck Tumor Management Conference
Weekly Wed., 4:00 P.M., Contact: Dr. Manning
Hematology-Oncology Clinical Conference
Weekly Tuesday, Noon-1 P.M., Contact: Sidney Salmon, M.D., Room 6505, Dept. of Internal Medicine.

Infectious Disease Conference
2nd Thursday, 12:00 Noon, TMC, Contact: Dr. B. Friedman.

Medical Conference
Weekly Wed., 8:00 A.M., TMC, Contact: Dr. M. Fuchs

Medical Grand Rounds
Weekly Wed., 12:00-1:00 P.M., AHSC, Contact: Dr. J. Smith.

Morbidity/Mortality in E.M.
2nd Tusday, 9:00 A.M., AHSC, Contact: Drs. Hughes & Alcorn

Nephrology Conference
4th Thursday, 12:00 Noon, TMC, Contact: Drs. Seltzer or Smithline

Neuromuscular Disease Conference
Weekly Friday, 11:30 A.M., Contact: Dr. Stern

Neuropathology Review Conference
Thursdays, 8:30 A.M., Rm. 5120, UAHSC.

Neuroradiology Conference
Weekly Thurs., 5:00 P.M., Contact: Dr. P.C. Christenson

Neuromuscular Journal Conference
2nd & 4th Thursday, 7:00-9:00 P.M., Contact: Dr. Stern.

Neuropathology Case Review
Weekly Friday, 8:30 A.M., AHSC, Contact: Dr. P. Johnson.

Neurosciences Seminar
Weekly Tuesday & Friday, 7:30 A.M., AHSC, Contact: Dr. C. Bamford

Nuclear Medicine
Weekly Thursday, 7:30 A.M., AHSC Radiology Conference Room

Inter-Hospital Nuclear Medicine Conference
Weekly Friday, 7:15 A.M., TMC, Contact: S. V. Hilts

OB/GYN Conference
1st Friday, 7:30 P.M., TMC, Contact: Dr. P. Evans

OB/GYN Pathology Conference
4th Friday, 1:30 P.M., AHSC, Contact: Dr. R. Spark

Ophthalmology Grand Rounds
Weekly Friday, 8:00 A.M., AHSC, Contact: Dr. H. Cross

Ophthalmology Retina & Fluoro. Conference
Weekly Thursday, 5:00 P.M., AHSC Contact: Dr. H. Cross

Orthopedic Conference
1st Tuesday, 7:30 P.M., TMC. Contact: Dr. L. Mann

Orthopedic Rounds
Saturday, 8:00 A.M., Contact: Dr. Peltier.

Pain Conference
3rd Monday, 4:00-5:00 P.M., AHSC Dining Rm. C&D, Contact: Drs. Hameroff & Cork.

Pathology Seminar
Weekly, 4:30-5:30 P.M., AHSC, Room 5120

Clinical Pathology Conference
5th Thursday, TMC, Contact: Dr. M. Fuchs

Tucson Pathologist Conference
1st Monday, 7:30 P.M., AHSC, Contact: Dr. A. R. Graham.

Pediatric Grand Rounds
Weekly Tuesday, 12:30-1:30 P.M., TMC & AHSC, Contact: Dr. H. Thompson.

Pediatric Problem Patient Conference
Weekly Wed., 8:00 A.M., Contact Dr. Walson

Pediatric Research Forum
Weekly Tuesday, 7:30 A.M., Contact: Dr. James Jones

Pediatric Specialty Conference
Weekly Friday, 8:00 A.M., Contact: Dr. Philip Walson.

Peri-Natal Conference
2nd Monday, 7:30 P.M., AHSC, Contact: Dr. J. Lohman.

Psychiatric Grand Rounds
Weekly Wed., 4:00 P.M., Contact: Dr. R. Cutts

Psychiatric Monthly Case Conference
2nd Friday, 7:30 A.M., Contact: Dr. Alan Levenson,Palo Verde Hospital

Pulmonary Rounds
Weekly Friday, 11:30 A.M., Contact Dr. Benjamin Burrowns.

Chest Radiology
Weekly Monday, 5:00-6:00 P.M., Rm 1535F, UAHSC. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.

Neuroradiology Conference
Weekly Thursday, 5:00 P.M., TMC, Contact: Dr. N. Komar

Neuroradiology Teaching Conference
Weekly Wednesday, 7:30 A.M., AHSC, Contact: Dr. Christenson

Radiation Oncology Planning Conference
Weekly Friday, 8:30-10:00 A.M., AHSC, Room 0655

Radiology Interesting Case Conference
Weekly Thursday, 12:00 Noon, AHSC, Contact: Dr. Freundlich

Gastrointestinal Radiology
Weekly Tuesday, 5:00-6:00 P.M., AHSC, Contact: Radiology Dept.

Radiology-Rheumatology Conference
Weekly Thursday, 7:45 A.M., UAHSC, Library Rm.1535C. **Uroradiology (Genitourinary Radiology)**
2nd & 4th Mondays, 7:30- 8:30 A.M., AHSC, Contact: Dept. of Radiology

Renal Pathology Conference
1st & 3rd Thurs., 11:30 A.M., Contact: Dr. Nagle.

Residents Noon Conference
Weekly Tuesday & Thursday, 12:00 Noon, AHSC, Contact: Dr. A. Greensher.

Resident's Conference
Wednesday, weekly 5-6 p.m., Diag. Radiology Conf. Rm.

Surgical Lit/Basic Skills
Weekly Friday, 1:00 P.M., TMC, Contact: P. Whitney

Surgical Morbidity & Mortality Conference
Weekly Wed., 8:00 A.M., Contact: Dr. Wangensteen.

Trauma Conference
Every Thursday,4-5 p.m. UAHSC, Rm. 5505.

Ward Grand Rounds - Trauma
Weekly Tuesday, 8:00 A.M., Contact: Dr. M. Silverstein.

Toxicology Conference
Weekly Tuesday, 8:00 A.M., Contact: Dr. Keith Likes
Section.

Tumor Conference
Weekly Thursday, 12:00 Noon, TMC, Contact: Dr. D. Neubauer

Tucson Ultrasonography Group
Weekly Wed., 4:30 P.M., AHSC, Contact: Dr. I. Freundlich.

General Urology Conference
Weekly Tuesday, 12:00 Noon, AHSC & VA Hospital Contact: Dr. G. W. Drach.

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Studies confirm that 9 out of 10 smokers know that smoking is a bad habit. And they'd like to quit. But they don't know how.

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The National Cancer Institute will provide the "Helping Smokers Quit" kit free of charge to all physicians who want to participate in this important effort. Included in the kit are guidelines for physicians, a self-test to help smoking patients determine why they smoke, pamphlets with tips on quitting, and waiting room posters to introduce the subject. Each kit contains enough materials to help 50 of your smoking patients who want to quit.

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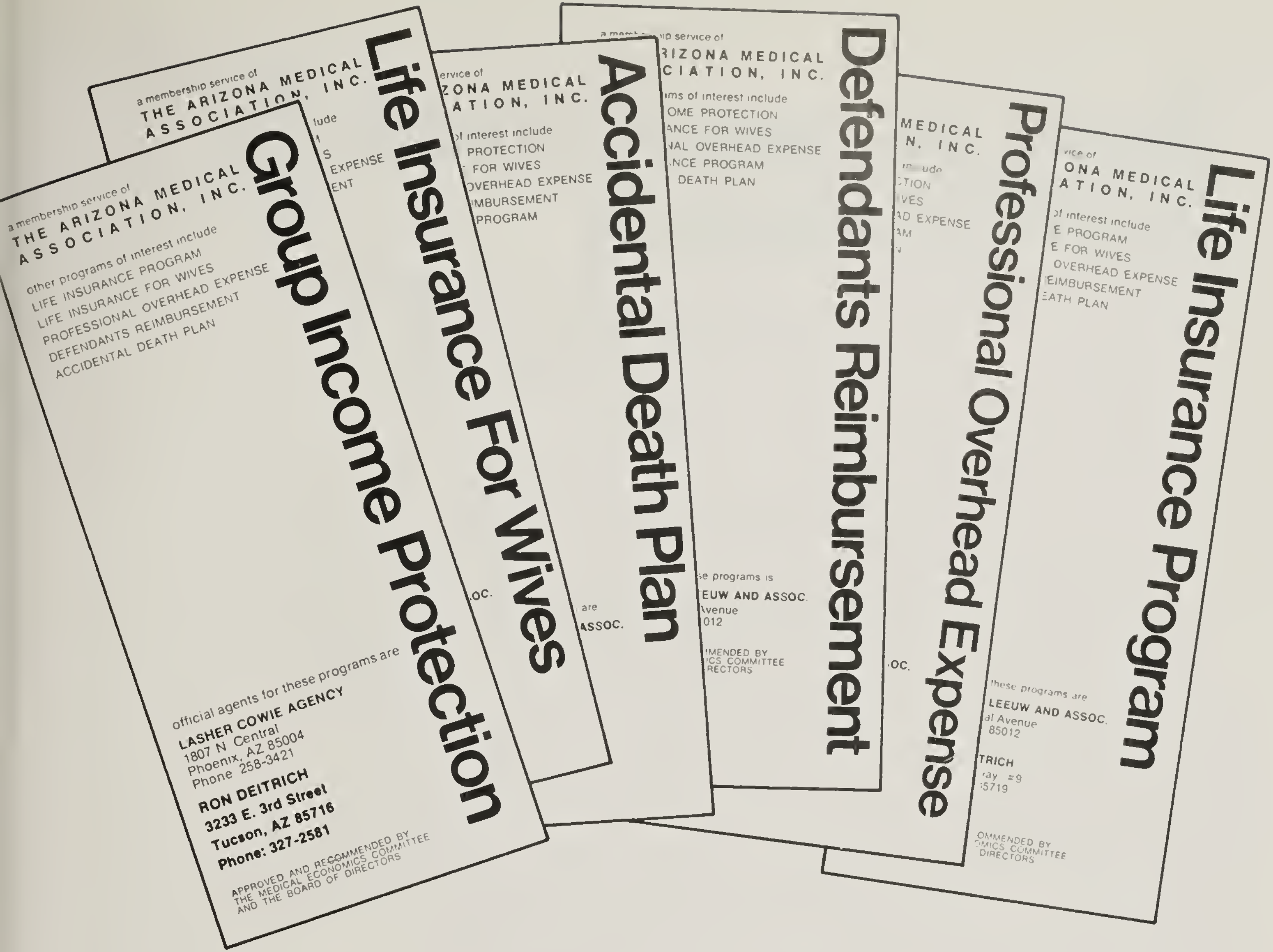

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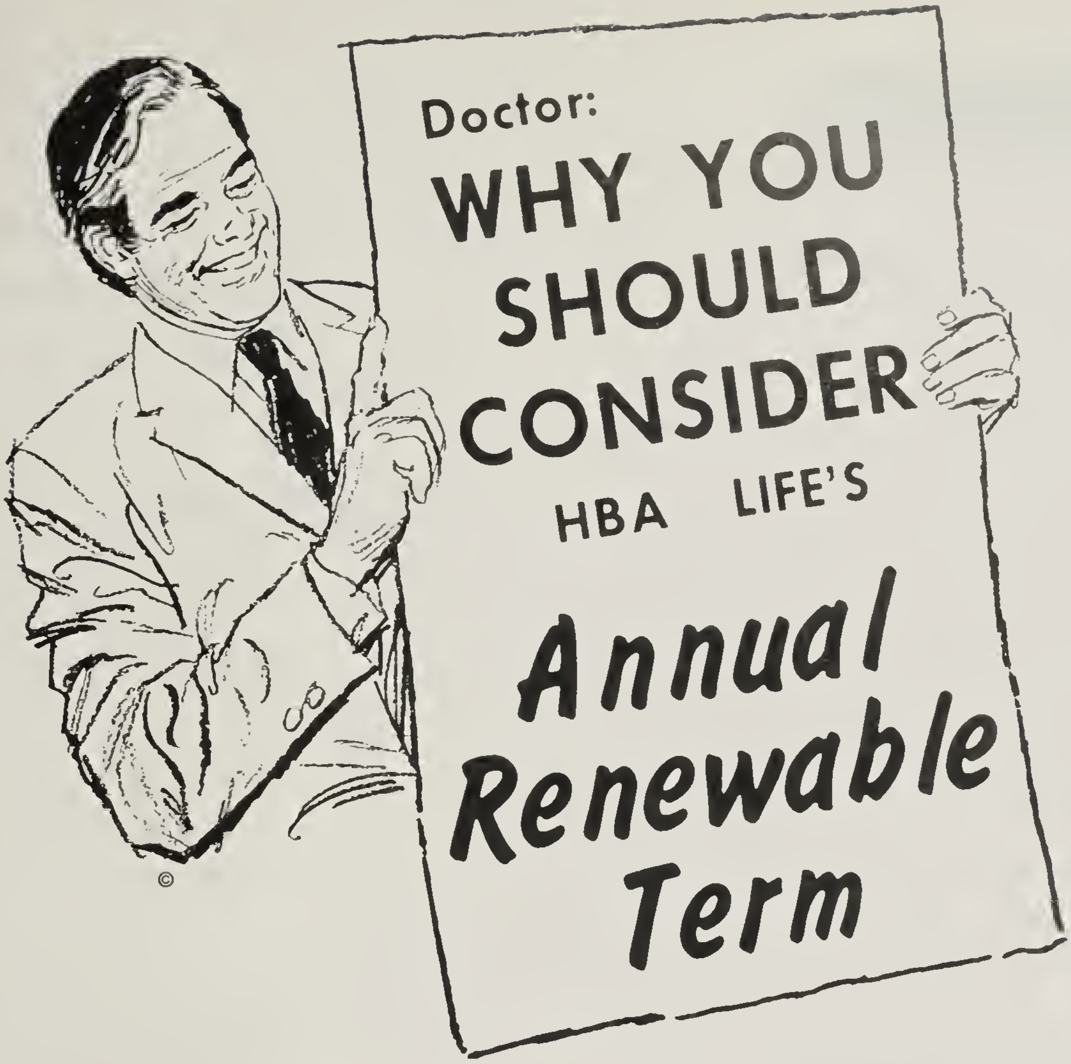

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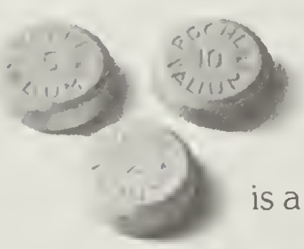

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Another important aspect of the clinical character of Valium is safety. Though drowsiness, ataxia and fatigue are possible, these and more serious side effects are rarely a problem. Of course, as with all CNS-acting drugs, patients taking Valium should be cautioned against driving, operating dangerous machinery or the simultaneous ingestion of alcohol.

Unquestionably, many psychotherapeutic agents, including other benzodiazepines, have antianxiety effects. But one fact remains: you get a certain kind of patient response with Valium. It's a response you want. A response you know. A response you trust as part of your overall management of anxiety and psychic tension.

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Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors, psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

The effectiveness of Valium (diazepam/Roche) in long-term use, that is, more than 4 months, has not been assessed by systematic clinical studies. The physician should periodically reassess the usefulness of the drug for the individual patient.

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

> *Usage in Pregnancy:* **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

Dosage: Individualize for maximum beneficial effect. *Adults:* Tension, anxiety and psychoneurotic states, 2 to 10 mg b.i.d. to q.i.d.; alcoholism, 10 mg t.i.d. or q.i.d. in first 24 hours, then 5 mg t.i.d. or q.i.d. as needed; adjunctively in skeletal muscle spasm, 2 to 10 mg t.i.d. or q.i.d.; adjunctively in convulsive disorders, 2 to 10 mg b.i.d. to q.i.d. *Geriatric or debilitated patients:* 2 to 2½ mg, 1 or 2 times daily initially, increasing as needed and tolerated (See Precautions.) *Children:* 1 to 2½ mg t.i.d. or q.i.d. initially, increasing as needed and tolerated (not for use under 6 months).

Supplied: Valium® (diazepam) Tablets, 2 mg, 5 mg and 10 mg—bottles of 100 and 500; Tel-E-Dose® packages of 100, available in trays of 4 reverse-numbered boxes of 25, and in boxes containing 10 strips of 10; Prescription Paks of 50, available singly and in trays of 10.

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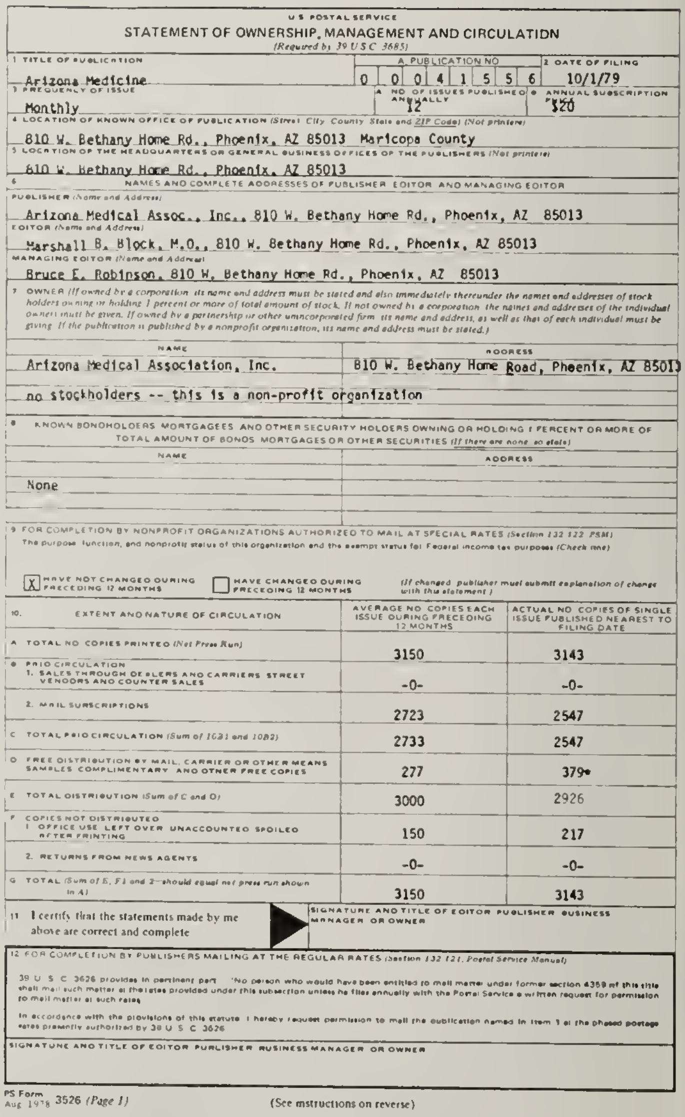

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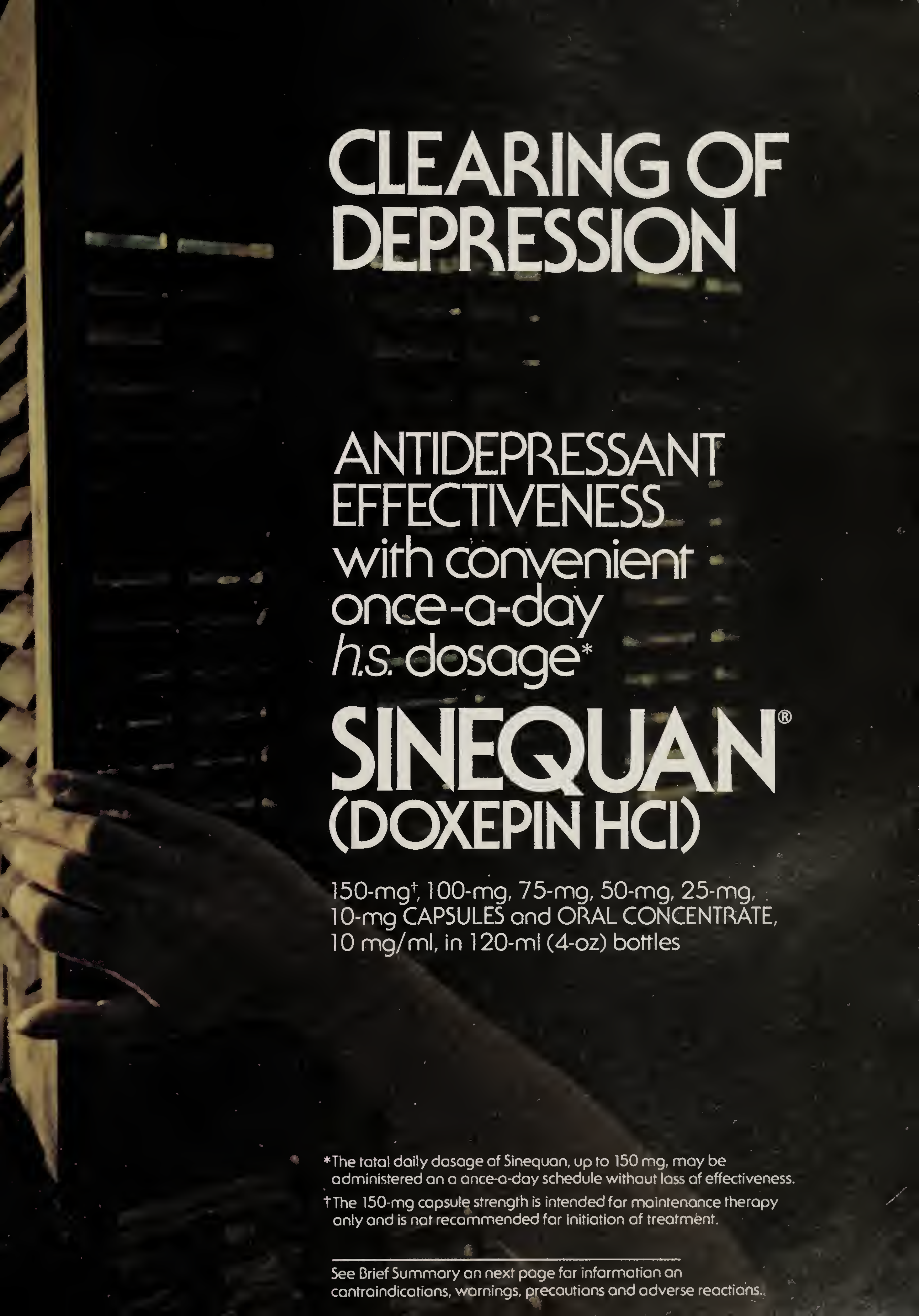

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†The 150-mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.

See Brief Summary on next page for information on contraindications, warnings, precautions and adverse reactions.

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which may help to relieve the difficulty in falling and staying asleep, and the early-morning awakening often associated with depression.

ESTABLISHED ANTIANXIETY ACTIVITY

to help alleviate the anxiety which often accompanies clinical depression.

USUALLY WELL TOLERATED

At doses up to 150 mg per day, Sinequan does not generally affect the antihypertensive activity of guanethidine and related compounds. Tachycardia and hypotension have been reported occasionally. Drowsiness is the most commonly observed side effect. Dry mouth, blurred vision, constipation and urinary retention have been reported.

EXTENDED RANGE OF DOSAGE STRENGTHS

for flexibility in individualizing therapy.

BRIEF SUMMARY
SINEQUAN® (doxepin HCl) Capsules/Oral Concentrate

Contraindications. Contraindicated in individuals who have shown hypersensitivity to the drug, and in patients with glaucoma or a tendency to urinary retention. These disorders should be ruled out, particularly in older patients Possibility of cross sensitivity with other dibenzoxepines should be kept in mind

Warnings. The once-a-day dosage regimen of SINEQUAN (doxepin HCl) in patients with intercurrent illness or patients taking other medications should be carefully adjusted This is especially important in patients receiving other medications with anticholinergic effects

Usage in Geriatrics: The use of SINEQUAN on a once-a-day dosage regimen in geriatric patients should be adjusted carefully based on the patient's condition.

Usage in Pregnancy: Reproduction studies performed in animals have shown no evidence of harm to the animal fetus Since there is no experience in pregnant women receiving this drug, safety in pregnancy has not been established There are no data with respect to the secretion of the drug in human milk and its effect on the nursing infant.

Usage in Children: Usage in children under 12 years of age is not recommended because safe conditions for its use have not been established

MAO Inhibitors: Serious side effects and even death have been reported following the concomitant use of certain drugs with MAO inhibitors Therefore, MAO inhibitors should be discontinued at least two weeks prior to the cautious initiation of therapy with this drug The exact length of time may vary and is dependent upon the particular MAO inhibitor being used, the length of time it has been administered and the dosage involved

Usage with Alcohol: It should be borne in mind that alcohol ingestion may increase the danger inherent in any intentional or unintentional SINEQUAN overdosage. This is especially important in patients who may use alcohol excessively

Precautions. Since drowsiness may occur with the use of this drug, patients should be warned of that possibility and cautioned against driving a car or operating dangerous machinery while taking this drug

Patients should also be cautioned that their response to alcohol may be potentiated

Since suicide is an inherent risk in any depressed patient, and may remain so until significant improvement has occurred, patients should be closely supervised during the early course of therapy Prescriptions should be written for the smallest feasible amount

Should increased symptoms of psychosis or shift to manic symptomatology occur, it may be necessary to reduce dosage or add a major tranquilizer to the dosage regimen.

Adverse Reactions. NOTE Some of the adverse reactions noted below have not been specifically reported with SINEQUAN use However, due to the close pharmacological similarities among the tricyclics the reactions should be considered when prescribing SINEQUAN

Anticholinergic Effects Dry mouth, blurred vision, constipation, and urinary retention have been reported If they do not subside with continued therapy, or become severe, it may be necessary to reduce the dosage

Central Nervous System Effects Drowsiness is the most commonly noticed side effect This tends to disappear as therapy is continued Other infrequently reported CNS side effects are confusion, disorientation, hallucinations, numbness, paresthesias, ataxia, and extrapyramidal symptoms and seizures

Cardiovascular Cardiovascular effects including hypotension and tachycardia have been reported occasionally

Allergic Skin rash, edema, photosensitization, and pruritus have occasionally occurred

Hematologic Eosinophilia has been reported in a few patients. There have been occasional reports of bone marrow depression manifesting as agranulocytosis, leukopenia, thrombocytopenia, and purpura

Gastrointestinal Nausea, vomiting, indigestion, taste disturbances, diarrhea, anorexia, and aphthous stomatitis have been reported. (See anticholinergic effects)

Endocrine Raised or lowered libido, testicular swelling, gynecomastia in males, enlargement of breasts and galactorrhea in the female, raising or lowering of blood sugar levels have been reported with tricyclic administration.

Other Dizziness, tinnitus, weight gain, sweating, chills, fatigue, weakness, flushing, jaundice, alopecia, and headache have been occasionally observed as adverse effects

Dosage and Administration. For most patients with illness of mild to moderate severity, a starting daily dose of 75 mg is recommended Dosage may subsequently be increased or decreased at appropriate intervals and according to individual response. The usual optimum dose range is 75 mg/day to 150 mg/day

In more severely ill patients higher doses may be required with subsequent gradual increase to 300 mg/day if necessary Additional therapeutic effect is rarely to be obtained by exceeding a dose of 300 mg/day

In patients with very mild symptomatology or emotional symptoms accompanying organic disease, lower doses may suffice Some of these patients have been controlled on doses as low as 25-50 mg/day

The total daily dosage of SINEQUAN (doxepin HCl) may be given on a divided or once-a-day dosage schedule. If the once-a-day schedule is employed the maximum recommended dose is 150 mg/day This dose may be given at bedtime. **The 150 mg capsule strength is intended for maintenance therapy only and is not recommended for initiation of treatment.**

Antianxiety effect is apparent before the antidepressant effect. Optimal antidepressant effect may not be evident for two to three weeks

Overdosage.

A. Signs and Symptoms

1 Mild Drowsiness, stupor, blurred vision, excessive dryness of mouth

2. Severe Respiratory depression, hypotension, coma, convulsions, cardiac arrhythmias and tachycardias.

Also: urinary retention (bladder atony), decreased gastrointestinal motility (paralytic ileus), hyperthermia (or hypothermia), hypertension, dilated pupils, hyperactive reflexes

B Management and Treatment

1 Mild Observation and supportive therapy is all that is usually necessary

2. Severe Medical management of severe SINEQUAN overdosage consists of aggressive supportive therapy If the patient is conscious, gastric lavage, with appropriate precautions to prevent pulmonary aspiration, should be performed even though SINEQUAN is rapidly absorbed The use of activated charcoal has been recommended, as has been continuous gastric lavage with saline for 24 hours or more. An adequate airway should be established in comatose patients and assisted ventilation used if necessary EKG monitoring may be required for several days, since relapse after apparent recovery has been reported Arrhythmias should be treated with the appropriate antiarrhythmic agent It has been reported that many of the cardiovascular and CNS symptoms of tricyclic antidepressant poisoning in adults may be reversed by the slow intravenous administration of 1 mg to 3 mg of physostigmine salicylate. Because physostigmine is rapidly metabolized, the dosage should be repeated as required. Convulsions may respond to standard anticonvulsant therapy, however, barbiturates may potentiate any respiratory depression. Dialysis and forced diuresis generally are not of value in the management of overdosage due to high tissue and protein binding of SINEQUAN

Supply. SINEQUAN is available as capsules containing doxepin HCl equivalent to: 10 mg, 75 mg, and 100 mg doxepin bottles of 100, 1000, and unit-dose packages of 100 (10 x 10's) 25 mg and 50 mg doxepin: bottles of 100, 1000, 5000, and unit-dose packages of 100 (10 x 10's) 150 mg doxepin: bottles of 50, 500, and unit-dose packages of 100 (10 x 10's). SINEQUAN Oral Concentrate (10 mg/ml) is available in 120 ml bottles with an accompanying dropper calibrated at 5 mg, 10 mg, 15 mg, 20 mg, and 25 mg Each ml contains doxepin HCl equivalent to 10 mg doxepin. Just prior to administration, SINEQUAN Oral Concentrate should be diluted with approximately 120 ml of water, whole or skimmed milk, or orange, grapefruit, tomato, prune or pineapple juice. SINEQUAN Oral Concentrate is not physically compatible with a number of carbonated beverages For those patients requiring antidepressant therapy who are on methadone maintenance, SINEQUAN Oral Concentrate and methadone syrup can be mixed together with Gatorade®, lemonade, orange juice, sugar water, Tang®, or water, but not with grape juice. Preparation and storage of bulk dilutions is not recommended

More detailed professional information available on request

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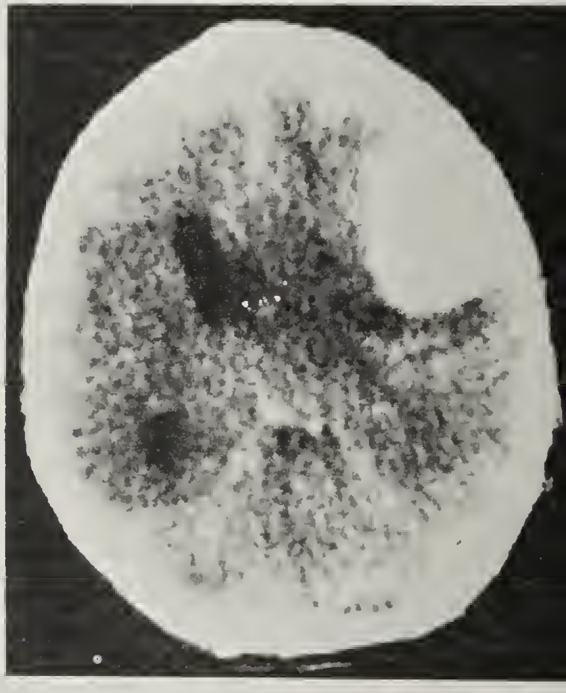

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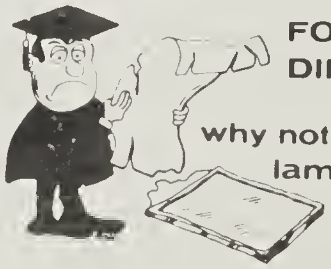

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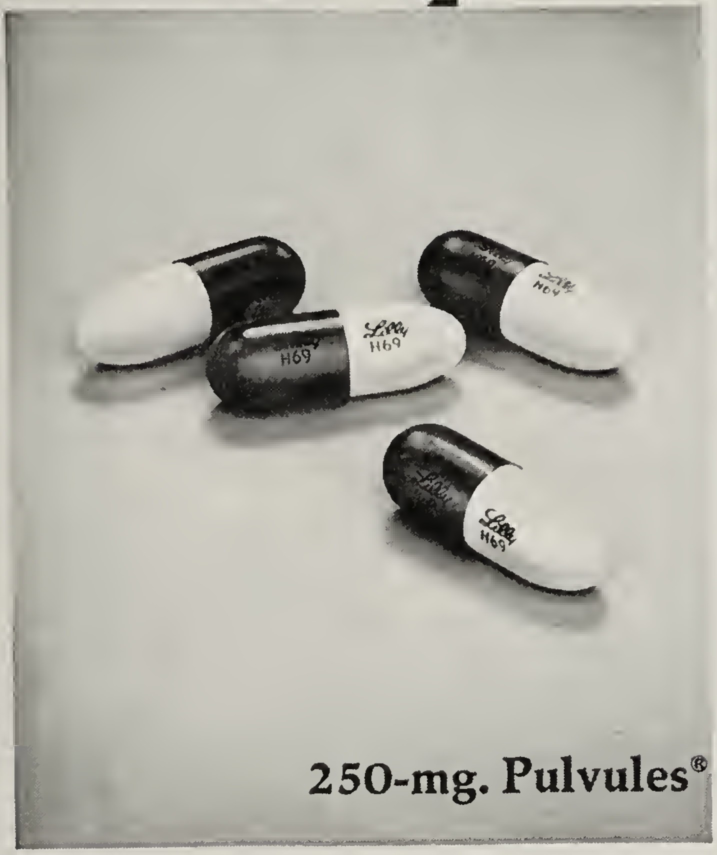
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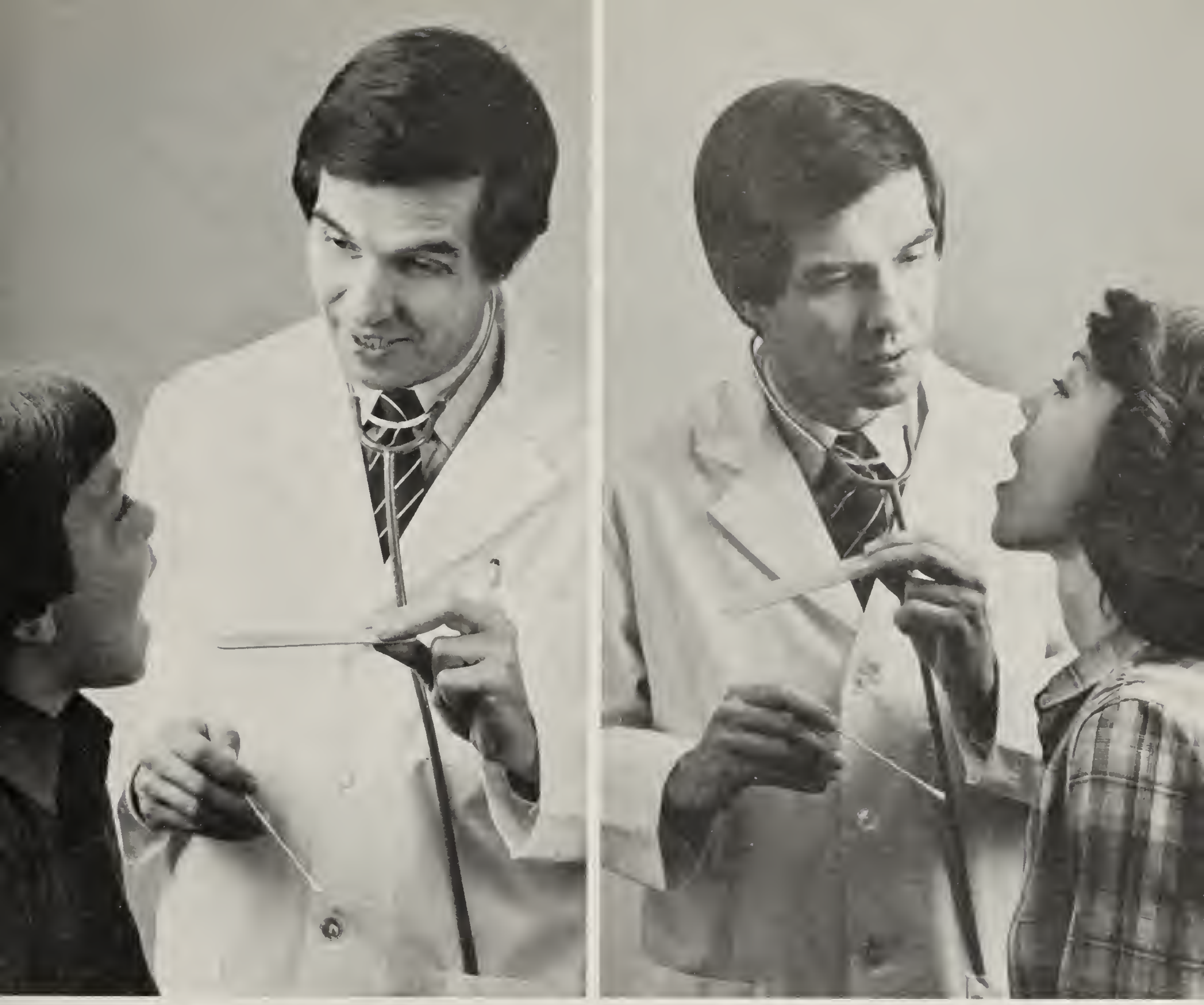

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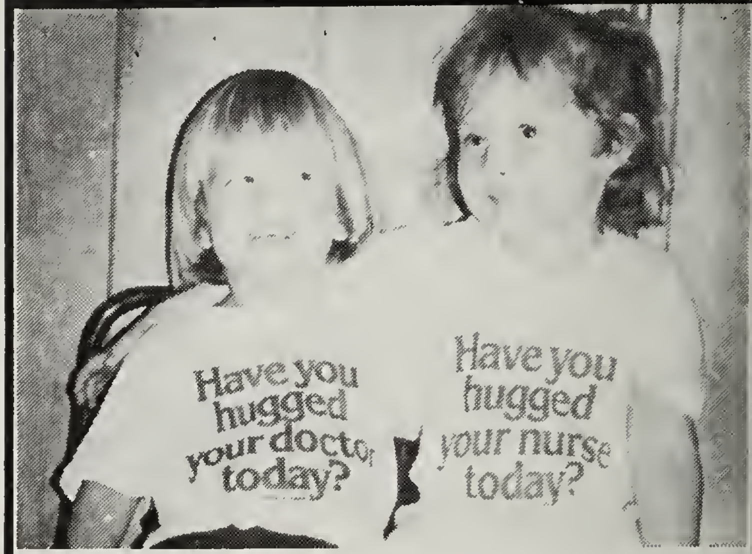
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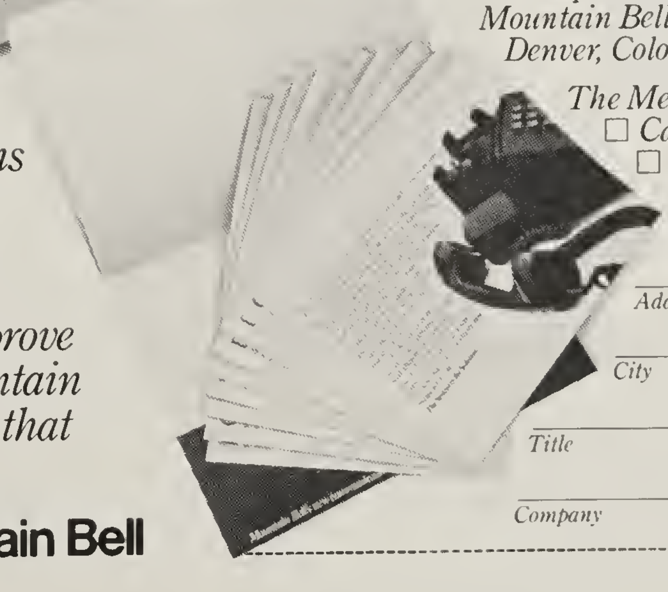

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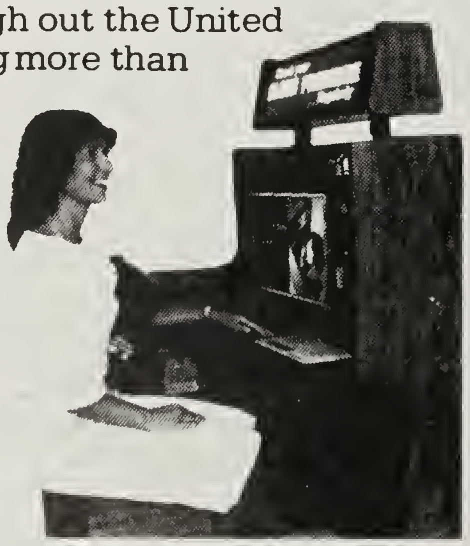

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Seminars in
Chest Medicine

Fat Embolism

Gerald L. Oldham, M.D.
William C. Weese, M.D.

Editors:
Faysal M. Hasan, M.D.
Richard I. Lemen, M.D.
Bernard E. Levine, M.D.

Fat embolism is a complex syndrome characterized by entrance of intracellular fat globules into veins and distribution through the pulmonary and systemic circulation. It is commonly associated with multisystem trauma, but exerts its greatest mortality from pulmonary complications. The purpose of this paper is to review etiologic theories of fat embolism and current therapeutic concepts.

Reports of animal experiments date to the 1600's, but the first description of fat droplets in pulmonary capillaries of accident victims was made by Zenker in 1862. Eleven years later, Von Bergmann reported the clinical triad of petechiae, dyspnea, and confusion. Not until 1964, however, was the severe degree of accompanying hypoxemia appreciated when Sproule, Brady, and Giblert measured arterial blood gases in three patients.[1] Subsequently, understanding of pathophysiology and treatment of fat embolism has advanced to its present state.

The reported incidence of fat embolism varies widely, depending upon the degree and type of injury, quality of medical and surgical care, accuracy of pathologic analysis, and length of survival.[2] It has been found in 90% of immediate deaths following trauma and fractures, in 100% of sudden violent deaths, and 80% of deaths with multiple compound fractures sustained in traffic accidents. The longer a patient survives, the fewer fat emboli are seen at autopsy; the highest incidence was found 48 hours after injury. Overall mortality rate from fat emboli due to multiple trauma is 5-15%.[3]

The various clinical conditions associated with fat embolism are listed in Table 1. Trauma, especially following long bone or pelvic fracture, is the most common antecedent event. In survivors with multiple or combined fractures, incidence of fat embolism increases to 20-30%. Highest prevalence (37%) occurs after severe burns. Common to all underlying events is either damage to fat-containing tissues or intravascular hypercoagulability. Age and sex are not determining factors. Fat embolism has been reported in children and even in neonate. Men are historically more afflicted because of increased occupational exposure to major trauma. Recurrence of fat embolism has not been reported.[3]

There are two commonly accepted theories regarding the source of fat emboli. The mechancial theory postulates an extravascular source caused by disruption of fat-containing tissue. It implies liberation of fat globules from either bone marrow or subcutaneous soft

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From: Dept. of Internal Medicine, St. Joseph's Hospital and Medical Center, Phoenix, Arizona. Dr. Oldham, (Resident in Medicine, 1976-1979), 13660 North 94th Ave, Ste. F-5, Peoria, Arizona 85345. Dr. Weese (Attending Physician), 550 West Thomas Road, Ste 112-B, Phoenix, AZ 85013.

Clinical States Associated with Pulmonary Fat Embolism*

Trauma, especially with fracture.
Extensive burns.
Post-abdominal surgery, especially with damage to the liver.
Post cardiac massage.
Post open-heart surgery with extracorporeal circulation.
Post orthopedic surgery—e.g. manipulative release of joint contractures, repair of club foot deformity.
Poisoning—e.g. carbon tetrachloride, phosphorus, mercuric chloride, chloroform, ethyl alcohol, aviation fuel inhalation.
Chronic alcoholism.
Diabetes mellitus with hyperlipemia.
Sickle cell anemia.
Sepsis.
Collagen vascular disease—e.g. dermatomyositis, rheumatoid arthritis.
Eclampsia: traumatic fetal extractions.
Chushingoid states.
Hemoglobinopathies.
Pancreatitis.
Rapid decompression.
Iatrogenic causes—e.g. overhydration, inadvertent intravenous milk injection, cerebral embolism post lymphangiography.

*Adapted from[3] [4] [5]

tissue, tearing of venules, and a force which absorbs or pushes the globules into the circulation.[6] The biochemical theory describes the intravascular formation of fat macroglobulins. It explains presence of fat emboli in conditions unrelated to trauma and states that fat droplets form in the circulation secondary to an alteration in lipid stability in response to injury initiated by tissue lipase, catecholamines, glucagon, and/or steroids.[7] A third theory, not as yet confirmed, suggests that fat macroglobulin may be formed by the release of free fatty acids from adipose tissue.[3]

Irrespective of the origin of fat embolism, certain clincial conditions contribute greatly to the development of the syndrome. On the systemic level are hemorrhagic shock (perhaps facilitating entry of fat droplets into the blood at the fracture site), disseminated intravascular coagulation (possibly triggered by fat emboli) and sepsis. Local factors (such as high concentration of proteases in hematomas at the fracture site and elevation of local tissue pressures) may also play a role. Three possible fates await the fat emboli in the lung. Small emboli (under 20 microns) may mould to the vessel caliber and pass through the lung to enter the systemic circulation and be either trapped in tissues or eliminated through renal glomeruli. Secondly, alveolar cells may engulf the fat particle, then desquamate into the alveolar space, and be eliminated through the sputum. Finally, there may be local lipolysis within the lung parenchyma, leading to chemical pneumonitis.[3] Free fatty acids release from hydrolysis of the fat droplets cause direct injury to the alveolar-capillary membrane with disruption of surfactant activity and interstitial edema, hemorrhage, and atelectasis.[7]

Clinically, there can be acute and delayed phases of pulmonary fat embolism. The former phase, rarely of significance, involves immediate occlusion of pulmonary capillaries, elevated pulmonary arterial and right atrial pressures, and cor pulmonale within 24 hours.[2] The more commonly manifest delayed effect occurs 24-72 hours after injury. Additionally, the fat droplets (with large surface areas) become coated with platelets; the resulting thrombocytopenia can lead to further pulmonary hemorrhage and petechiae over the chest, flanks, axilla, soft palate, and conjunctiva. Sequestered platelets release serotonin and 5-hydroxytryptamine, respectively precipitating bronchospasm and vasospasm.[6]

Clinical features of fat embolism include trachycardia, tachypnea, fever, petechiae, and neurologic deficit.[8] Symptoms which usually appear within 24-48 hours may be delayed for up to four days. A petechial rash is present in 25-50% of cases and is considered very suggestive of fat embolism.[5] Neurologic signs (ranging from confusion and restlessness to stupor and frank coma) may be due to ischemia from the fat emboli or from peripheral hypoxemia. Weisz et al., reviewing 31 deaths from pulmonary fat embolism, found only one patient with sufficeint cerebral fat to explain the fatal outcome. The authors conclude that systemic fat embolism was, in essence, a respiratory disease.[4]

Chest x-ray may remain normal, but often shows diffuse alveolar and interstitial infiltrates, most prominent at the bases. Pleural effusion is absent. The roentgenographic abnormalities usually clear within 7-10 days. Ventilation-perfusion lung scans and pulmonary arteriography are rarely of diagnostic value.[5] Electrocardiogram is usually within normal limits or shows non-specific changes.

Most significant laboratory data are the arterial blood gases; hypoxemia is almost always present and may be severe despite oxygen supplementation. Hemoglobin levels fall an average of 4-5 gm/100 ml.[9] Platelet count is less the 150,000/mm^3 in 80% of patients.[5] Presence of fat globules in the urine is not diagnostic, but its absence weighs against the diagnosis of fat embolism. Variable elevations of serum lipase are present and hypocalcemia, prolonged partial thromboplastin time, and decreased fibrinogen levels may also be found.

Prior to the mid-1960's, treatment was aimed primarily toward prevention of fat emboli and their systemic effects. Intravenous 5% ethyl alcohol was used to inhibit lipoprotein lipase, with one report showing a significant decrease in incidence of fat embolism in patients whose blood alcohol levels exceeded 0.03 gm% at the time of injury.[10] Heparin has also been employed to inhibit platelet aggregation and therefore to block release of serotonin and 5-hydroxytryptamine, but increased the risk of hemorrhage. Also popular were low molecular weight dextran (to improve capillary blood flow) and carbon dioxide inhalation (to dilate cerebral arteries and increase perfusion).

Treatment in recent years has emphasized reversal of effects of the fat emboli on the lungs and central nervous system. The goals are correction of hypoxemia (with ventilatory support, if necessary), maintenance of a negative but adequate fluid balance, and corticosteroids. ntubation and mechanical ventilation with positive and expiratory pressure (PEEP) has become a standard procedure in the face of severe hypoxia. PEEP increases unctional residual capacity and helps to decrease atelectasis and right-to-left pulmonary shunting of blood. Oxygenation is thus enhanced and the risk of oxygen toxicity is lessened.

Ashbaugh and Petty reported benefits from corticosteroids in 1966,[2] and their findings have been since confirmed.[8,9] In conjunction with the above supportive measures, there would often be mprovement in oxygenation within 12-24 hours, in the chest roentgenogram within 24-48 hours, and in neurologic status within 48-72 hours. The efficacy of corticosteroids is probably due to multiple actions: reduction in inflammatory effect of fatty acids on alveolar-capillary membranes, decrease in edema, stabilization of lipid membranes, increase in vascular ntegrity, and improvement in ventilation-perfusion ratios by combating the bronchospastic effect of serotonin and the vasospastic effect of 5-hydroxytryptamine.[6]

Summary

Fat embolism may present as a rapidly fatal illness manifest by the adult respiratory distress syndrome and marked neurologic deficiency. Pulmonary damage is mechanical from the emboli themselves and/or chemical secondary to toxic effects of fatty acids and platelet release of serotonin and 5-hydroxytryptamine. Diagnosis is suspected by the clinical setting—usually trauma—with respiratory distress, hypoxemia, and often petechiae. Treatment is basically supportive, directed toward improvement in oxygenation and optimization of fluid balance. Corticosteroids help decrease the severity and duration of the syndrome.

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AM Seminars in Dermatology

Erythema Nodosum and Coccidioidomycosis

Duane C. Whitaker, M.D.
Peter J. Lynch, M.D.

Editor:
Robert A. Schwartz, M.D.

Erythema nodosum is a localized inflammatory disease of the skin and subcutaneous tissue. It is characterized by the appearance of multiple painful nodules, two or more centimeters in diameter, over the anterior portion of the lower legs. Swelling of the legs and arthralgia may also be present. Individual lesions come and go over a period of one to two weeks; the entire course of the disease is usually less than two months. Women are affected considerably more often than are men.

Erythema nodosum may occur as an idiopathic problem or may be associated with medication reactions (particularly birth control pills), inflammatory bowel disease, pregnancy, sarcoidosis, and a variety of infections such as tuberculosis, lymphogranuloma venereum, streptococcal disease, Yersinia enterocolitis, histoplasmosis, and coccidioidomycosis.

From: Section of Dermatology, Department of Internal Medicine, University of Arizona, Health Sciences Center, Tucson, AZ 85724.

In the southwestern United States coccidioidomycosis is the most common of the discernable causes for erthema nodosum. In fact, Smith in 1940 stated that in endemic areas of California, 95% of patients wtih erythema nodosum had coccidioidomycosis.[1] It seemed unlikely to us that, in spite of the endemic nature of coccidioidomycosis in southern Arizona, this figure would be representative of our clinical practice. Thus we elected to look at a more recent series of cases from the University of Arizona Health Sciences Center.

Methods

A diagnosis of erythema nodosum was made on 22 occasions in the Dermatology Clinic at the University of Arizona Health Sciences Center between January 1, 1974 and January 1, 1979. The charts for these 22 patients were reviewed. A diagnosis of erythema nodosum was accepted based on the clinical impressions of the examining dermatologist. A diagnosis of coccidioidomycosis was made when standard criteria such as those of the Arizona Thoracic Society could be satisfied.[2] Patient histories were accepted regarding past status of coccidioidin skin tests. Tube precipitin and latex particle agglutination tests were used for serological testing and skin tests were preformed using coccidioidin 1:100 antigen.

Results

Twenty-two patients with erythema nodosum were seen during the five year period from 1974 to 1979. There were 17 women and 5 men. This ratio of women to men corresponds to the usual proportion of women found in other series of erythema nodosum. All but four of the patients were white. In six of the 22 patients (27%) a diagnosis of coccidioidomycosis could be confirmed. The skin tests, serologic, radiologic, and demographic data for these six patients are summarized in the table. In three of the remaining 16 patients (14%) erythema nodosum was believed to be due to birth control pills or to pregnancy. One additional patient had concomitant streptococcal infection. No cause was found for the erythema nodosum in the remaining twelve patients. We suspected coccidioidomycosis in several of these patients with idiopathic disease, but either the laboratory tests were negative or, in three cases, the patient failed to return for follow-up evaluation.

Only one of the six patients with coccidioidomycosis gave a history of a significant upper respiratory disease. Thus, with the exception of their skin lesions, the remaining five patients had essentially asymptomatic coccidioidomycosis.

The diagnosis of coccidioidomycosis in these patients was made on the basis of laboratory findings (see table). Five of the six patients with coccidioidomycosis had positive coccidioidin skin tests. These five patients had indicated that previously applied skin tests had been negative. The sixth patient was not skin tested. Serologic tests for coccidioidomycosis were positive in three of the five patients so tested. No other laboratory tests (e.g. CBC, differential count, sedimentation rate, etc.) were helpful for distinguishing those patients with erythema nodosum secondary to coccidioidomycosis from the other patients with erythema nodosum. The chest radiogram showed changes consistent with coccidioidomycosis in three of the six patients.

Table 1
Demographic and laboratory data for patients with erthema nodosum and coccidioidomycosis

Patient	Sex	Age	Race	Skin Test Conversion	Serology*	Chest Radiogram
1	F	30	White	Pos	Neg	Abnormal
2	F	17	White	Pos	Pos	Neg
3	F	27	White	Pos	Pos	Abnormal
4	F	75	White	PT did not return for reading	Pos	Abnormal
5	F	20	White	Pos	Neg	Neg
6	F	24	White	Pos	Not done	Neg

*Positive tests indicate the presence of IgM precipitating antibody to coccidioidin as measured by the tube precipitin or latex particle agglutination test. Conversion was based on patient history of negative test within the last 2 years.

**"Abnormal" indicates that the radiologist believed that the changes present were consistent with a diagnosis of cocidioidomycosis.

The erythema nodosum resolved spontaneously during the second month in four of the six patients with coccidioidomycosis. One of the remaining two patients was lost to follow-up and the final patient had recurring lesions over an eight month period of time.

There was no evidence of dissemination of disease in any of the patients with coccidioidomycosis; apparent spontaneous resolution of their infection occurred in all of the patients.

Discussion

No less than 27% of our patients with erythema nodosum had concomitant coccidioidomycosis. All of our patients with these two diseases were women. Women in general[3] and women with coccidioidomycosis[4] seemed predisposed to develop erythema nodosum.

Erythema nodosum is generally believed to be a manifestation of hypersensitivity responsiveness in patients with coccidioidomycosis.[1, 5] For this reason skin tests are usually positive at the time when erythema nodosum lesions appear.[1, 5] This certainly appeared to be true for our patients since all five of the patients who could be evaluated had positive skin tests at the time the diagnosis of coccidioidomycosis was made. It should be noted, however, that a related skin disease, erythema multiforme, may precede skin test positivity in patients with coccidioidomycosis (unpublished observations).

loreover, erythema nodosum may appear prior to evelopment of skin test positivity in patients who have ssociated tuberculosis.[6] These two observations ogether with the occasional development of severe skin est reactions[7] suggest to us that serologic tests might est be carried out first. Thus if IgM precipitating ntibodies are present (a positive test), skin testing ould not have to be performed. Unfortunately, recipitating antibodies disappear rapidly and thus erologic tests may be negative by the time the patient ppears in the doctor's office. A negative serologic test hen must be followed by skin testing. All of our patients ere skin tested with coccidioidin 1:100. Spherulin, hich may detect a higher proportion of patients with occidioidomycosis, was not used, but because of this reater sensitivity it is our intent to apply both skin tests the future evaluation of seronegative patients with rythema nodosum.

Erythema nodosum is generally regarded as ortending a favorable prognosis in patients with occidioidomycosis; this was true for our six patients. lowever, this sign is not absolute as instances are nown where dissemination of the coccidioidomycosis ccurred in patients who had had erythema nodosum.[8]

Our six patients with erythema nodosum and occidioidomycosis were treated with aspirin, elastic vrap compression and, when necessary, bed rest. Corticosteroids were not found to be necessary and vere avoided on the theoretical grounds that they might ncrease the risk of dissemination. However, in spite of his concern no definite risk can be shown for patients so reated.[8] We would not hesitate to use the orticosteroids if necessary even as we occasionally do in reating erythema multiforme when it occurs in ssociation with coccidioidomycosis.

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Seminars in Endocrinology and Metabolism

Acute Renal Failure

After Intravenous Pyelography in Patients with Diabetes Mellitus

David Berger, M.D.

Editor:
Stephen E. Dippe, M.D.

Intravenous pyelography is widely used in the evaluation of renal disease in diabetic patients. Diabetic glomerulosclerosis, arteriolar nephrosclerosis, urinary tract infections, and papillary necrosis are some of the complications that may warrant the need for excretory urography. Although currently used radiographic contrast agents are considered safe in diabetics a number of cases have recently been reported in which acute renal failure followed intravenous pyelography.[1-6] The illustrative case presented below is followed by a review of the literature.

A 69-year old hypertensive diabetic man with background retinopathy, on insulin for 20 years,'was admitted to the hospital complaining of chest pain. Serial cardiac enzymes and the electrocardiograms were normal except for the presence of PVC's. The patient was digitalized due to mild congestive heart failure.

On the third hospital day an intravenous pyelogram was performed because of elevated blood urea nitrogen (BUN) and serum creatinine and 2+ proteinuria. The patient received a 50cc bolus of diatrizoate meglumine and diatrizoate sodium (Hypaque - 75) following a castor oil prep and overnight fluid restriction. There was no immediate adverse reaction such as rash, hypotension or cardiac arrhythmia; the study revealed bilaterally small kidneys with reduced function.

The patient was discharged on the fifth hospital day, but because of nausea, vomiting and weakness was re-admitted to the hospital four days later. Physical examination was unremarkable except for signs of dehydration, postural hypotension and retinopathy. The course of his renal function and pertinent lab data are depicted in the Table.

Therapy included fluid challenge with normal saline, Kayexalate enemas, insulin and careful monitoring of vital signs and urine output. The patient improved and a diuresis occurred; he was discharged six days later.

From: 555 West Catalina Drive, Phoenix, AZ 85013.

Clinical Data

	Before Study	Days After IV				
		1*	6	8	11	2
Bun, mg/dl (8 - 25)	42	63	63	34	28	2
Serum creatinine, mg/dl (0.6 - 1.5)	2.3	4.1	3.2	2.5	2.0	1.
Serum potassium, mEq/liter (3.5 - 5.0)	3.8	4.4	6.7+	4.8	4.7	4.
Serum carbon dioxide, mEq/liter (24 - 32)	24	20	24	29	29	2
Serum urates, mg/dl (3.8 - 7.1)	5.6	—	7.8	5.4	—	6.
Serum phosphate, mg/dl (2.5 - 4.5)	3.5	—	4.7	—	—	3.
Fasting blood sugar, mg/dl (65 - 110)	216	121	—	51	75	10
Serum digoxin level, ng/dl (0.5 - 2.0)	—	—	3.3	2.7	0.6	–

*Patient discharged on day 2 and re-admitted on day 6.
+Potassium exchange resin therapy being used.

Deterioration in renal function following intravenous pyelography in diabetics is not uncommon; over 60 cases have been reported to date.[1-7] The incidence of contrast media-induced renal failure is much higher if one considers not only urography but celiac arteriography, coronary angiography and cholecystrography, all of which have been shown to cause acute renal failure in diabetics.[7,8] While most patients recover spontaneously,[1-8] a number of cases of irreversible renal failure[5,9] and recently one death[10] have occurred following intravenous pyelography.

There are a number of factors which predispose diabetics to developing acute renal failure after urography. Age and duration of diabetes are important; the majority of patients are over 55 years of age and approximately 90% have had diabetes for more than 10 years.[4] The diabetic patients have a high prevalence of complications such as arterial hypertension, retinopathy and neuropathy.[3] Most have some degree of renal insufficiency, with or without proteinuria before urography (serum creatinine ≧ 1.5 mg/dl).[2-4,6,7] The use of diuretics and mild dehydration prior to study are also characteristic of many diabetics who develop this syndrome.[6] It appears though that the two major risk factors are the age at onset of diabetes and the serum creatinine level at the time of intravenous pyelograph.[5]

The clinical course of these patients is relatively uniform. Renal deterioration or oliguria occurs within 24 hours of the procedure and persists an average of 3 to 4 days.[2-8] It is important to note that in one series most patients did not become oliguric but simply experienced an increase in serum creatinine.[5] The serum creatinine, which rises by at least 1 mg/dl usually reaches its peak value within 5 days after urography.[2-8] Complete recovery is common and occurs in less than 14 days,[3-7] however, a minority of patients require dialysis; in this group some have suffered permanent impairment and need chronic dialysis.[5,11] With rare exception,[11] patients who develop irreversible renal damage have a pre-study serum creatinine in excess of 5 mg/dl.[3,5,6,8]

There is no known effective treatment of intravenous contrast media-induced acute renal failure. Furosemide, mannitol and intravenous fluids have been used with little success.[3,5] It is important to maintain adequate hydration prior to urography, thus avoiding the volume depletion that can occur with cathartics and overnight fluid restriction. However, several cases of oliguric renal failure have occurred in diabetics despite intravenous hydration before urography.[2,3,5,8,11] If the patient is dehydrated following study, fluids should be administered cautiously.[11] It is recommended in diabetics undergoing intravenous pyelography or another contrast study that both the urine output and serum creatinine should be monitored for the first 5 days.[3,4,6]

There have been many theories as to the mechanism of contrast media-induced renal failure in diabetes. As mentioned previously, dehydration prior to study is not a significant risk factor.[5,7,8] Similarly, hypotension during intravenous pyelography has not been shown to play a prominent role in the development of this type of renal faliure.[5] There has been no correlation between the occurrence of renal failure and the dose or type of contrast media utilized (salts of diatrizoic acid are standard).[5] Renal damage can arise with as little as 30cc of radiographic dye given as a bolus or by drip-infusion.[8] The combination of severe vascular disease and proteinuria have been implicated in the genesis of renal injury following urography.[9,11] Acute intrarenal obstruction and oliguria are postulated to occur secondary to reduced renal blood flow and subsequent precipitation of contrast material with urinary mucoprotein. However, this theory has not been well substantiated; a number of patients with only trace or no proteinuria have developed significant renal insufficiency following urography.[3,8,13] The uricosuric effects of contrast media have been mentioned as playing a role in the dye-induced renal failure,[14] however, normal urinary uric acid excretion before and after study in one diabetic militates against the uricosuria theory.[12] The currently accepted mechanism of contrast media-induced renal failure in diabetes relates a combination of factors. Contrast media may have direct vascular toxicity (renal vasoconstriction and cortical ischemia) and in patients with already compromised renal function characterized by decreased glomerular filtration rate prolonged exposure of the drug results.[3,5,6,8] Aggregation of erythrocytes and possibly platelets, which has been observed following injection of radiopaque agents leads to slowing of capillary flow and sludging.[15] These phenomena presumably cause ischemia and oliguria. This theory however is still only speculative.

Urography and other intravenous radiographic dye studies should not be done indiscriminantly in patients with diabetes mellitus. Patients with multiple myeloma are at risk for developing renal failure; diabetics of long duration and whose serum creatinine is greater than 1.5 mg/dl are especially prone to renal injury following administration of contrast media. These studies are dangerous and may precipitate irreversible renal failure when the patient's creatinine level is over 5 mg/dl. If urography is indicated (e.g. to R/O) obstruction secondary to calculi, tumor or papillary necrosis or investigate chronic urinary tract infection) the patient should be monitored closely for signs of renal deterioration afterwards.

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Seminars in Gastroenterology and Liver Disease

Primary Biliary Cirrhosis

Stephen Glouberman, M.D.

Editors:
George Burdick, M.D.
Stephen Glouberman, M.D.
Leon Rigberg, M.D.

Primary biliary cirrhosis is an uncommon, progressive hepatic disorder which usually results in a fatal outcome. About 90% of patients affected are women, and the disease usually has its onset between ages 40-59, although women in their 20's have been affected. The disease is characterized by progressive destruction of septal and interlobular bile ducts as a chronic nonspecific, nonpurulent destructive cholangitis, with the ultimate development of cirrhosis.

The etiology of this disorder is unknown. Some of the clinical and histologic features are suggestive of chronic active hepatitis, but there is no evidence that one of the hepatitis viruses is instrumental in the histopathogenesis. There is a potent immunologic response in primary biliary cirrhosis with elevated IgM and antimitochondrial antibodies being very useful diagnostic findings. Immune complexes are found in high concentration with activation of the complement system. It has been speculated that these are induced by antigens released from injured bile duct epithelia.[1] Their etiologic role, however, is unclear.

Pathology

Grossly, the liver is enlarged, smooth and greenish in color. As the disease progresses, cirrhosis develops and

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the liver bcomes nodular. Portal hypertension ensues with the development of splenomegaly and varices. Enlarged lymph nodes appear in the porta hepatis. At all stages of the disease, the grossly visible bile ducts and common bile duct are patent.

The histology of primary biliary cirrhosis is conveniently categorized into four stages, but more than one stage can be present in a patient at a given time. If a wedge biopsy is obtained, it is possible that more extensive disease will be seen than if a needle biopsy specimen is examined because of the larger amount of tissue available.

In *stage 1*, septal and larger interlobular bile ducts are damanged and surrounded by a dense infiltrate of lymphocytes, plasma cells, large histiocytes and a few eosinophils, This is the florid duct lesion and is considered to be pathognomic.[2] There may be lymphoid aggregates, with or without germinal centers, and granulomas with giant cells are located near a damaged duct in the portal tract. The ductular cells are swollen, proliferated, crowded and may rupture. Centrolobular cholestasis may occur. In *stage 2*, the involvement is more widespread, with acute and chronic inflammation visible and some fibrosis is seen. Interlobular ducts are significantly decreased in amount by about 60%, but small ductular proliferation occurs in the protal tracts. The granulomas are less frequently seen. There is hepatocellular necrosis in periportal locations, swelling and cholestasis. There may be piecemeal necrosis reminiscent of chronic active hepatitis. *Stage 3* consists of scarring with acellular septae extending from the protal tracts into and around the lobules. There is an occasional lymphoid aggregate, but overall inflammation is diminished. Periportal cholestasis may be severe. Cirrhosis with regenerative nodules is the hallmark of *stage* 4. There is a severe paucity of bile ducts and an occasional lymphoid aggregate may still be present. Mallory bodies may be present at any stage and is seen in 25% of patients, usually adjacent to a portal area.

Clinical Findings

The reason for the marked predominance of females in this disease is unknown. The condition may become evident in the third trimester of pregnancy, continue after delivery or abate and recur at a later time. Birth control pills may unmask the disorder.

Pruritus is the first symptom and may be present prior to the development of jaundice. The skin can become thickened or excoriated from scratching. A pigmentation may develop. Xanthomas can develop on extensor surfaces, and xanthelasma may appear on the eyelids. These tend to disappear preterminally. They can affect nerves resulting in a painful neuropathy. These are manifestations of elevated lipid levels, yet coronary artherosclerosis is usually not a clinical problem.[2] Collagen diseases, rheumatoid arthritis and the CRST syndrome are occasionally associated conditions. The sicca syndrome (dry eyes and mouth with or without the arthritis of Sjogren's syndrome) are present in 70% of patients.[2] Autoimmune thyroiditis and renal tubular acidosis may be present.

Diarrhea may be a problem and is related to steatorrhea. Vitamins A, D and K are malabsorbed. The vitamin D malabsorption results in osteoporosis and osteomalacia, with associated backache, rib pain and occasional pathologic fractures. Duodenal ulcer with hemorrahge can occur.

The liver is enlarged and portal hyertension with palpable splenomegaly and esophageal varices occur as the disease progresses. The liver disease is progressive and death ensues 6.6 years, on the average after symptoms devleop[3] from liver failure or ruptured varices.

An increasing number of patients are being found who are asymptomatic, despite having laboratory and histologic evidence for primary biliary cirrhosis. These patients are somewhat younger than the symptomatic ones, and tend to have less severely abnormal liver function tests. There is no correlation between histologic stage of the disease and the presence of symptoms, as 43% are found to be in stages 3 & 4. Four of five patients remained asymptomatic with stage 4 disease for 60 months, and some patients may remain asymptomatic for at least ten years.[5] The mean duration of the asymptomatic period, however is 2.2 years.[5]

Laboratory Findings

Anemia is often present and may be due to the liver disease, hypersplenism or gastrointestinal hemmorrhage. Liver function tests are abnormal with mild increases in transaminase and progressive decreases in albumin as the disease continues. The alkaline phophatase is very high, 5-10 times normal, or greater, and may be the clue to the diagnosis when found on blood sampling. The bilirubin is elevated in 69% of patients when the diagnosis is first made,[3] but may remain at normal levels for at least 10 years.[5] When the bilirubin starts to increase by 2 mg percent over a 6 month period of time, the disease is thought to have entered an accelerated phase and the mean length of survival thereafter is 49 months.[3] The antimitochondrial antibody test is of key diagnostic importance being found elevated in up to 96% of patients.[2] The diagnosis must be strongly entertained if this test is positive. Smooth muscle antibodies are present in 50% of patients and positive antinuclear antibodies in 5%. Calcium levels may be depressed because of fat malabsorption, and cholesterol and total lipid values are often greatly increased.

Diagnosis

The diagnosis of primary biliary cirrhosis is made by an elevated alkaline phophatase, a positive antimitochondrial antibody test and the hepatic histology. The large bile ducts are normal and their patency is shown by intravenous, transhepatic or retrograde cholangiography.

Treatment

The treatment is largely supportive. Cholestyramine, a bile salt binding resin, may alleviate pruritus and has also been associated with improvement in xanthomas. Supplemental Vitamins A and E can be given to support the low levels seen in primary biliary cirrhosis. Oral or parenteral vitamin D may not correct the bone changes. The involved liver may not be able to hydroxylate vitamin D and render it metabolically active. Giving 25, OH vitamin D can bypass this defect and result in an increase in the linear absorption coefficient of bone.[6]

These therapies do not overcome the steady progression of the disease. Because of immunologic phenomena present in primary biliary cirrhosis such as the antimitochondrial antibody levels, elevated IgM, and immune complexes, steroids and Azathioprine have been employed but without symptomatic or histologic benefit. Recently, d-penicillamine has been employed, and testing of this drug in primary biliary cirrhosis continues. The rationale for its use is its prevention of the formation of cross-linkage of collagen during its extracellular deposition, and its effect on depletion of hepatic copper. Copper accummulates in the liver during the disease and is known to be hepatotoxic. There appears to be a decrease in alkaline phosphatase and SGOT with d-penicillamine and an increase in albumin. The histologic staging is not improved. There does not appear to be an improvement in survival of symptomatic patients with 3 of 50 patients dying while on the drug, while 11 of 50 died with receiving placebo.[4] There is no significant effect on the course of asymptomatic patients. Twenty percent have to stop the drug because of side effects of nausea, vomiting. urticaria and proteinuria.

Prognosis

Primary biliary cirrhosis is considered to be a disease "that ultimately kills its victims,"[3] with death occuring 6.6 years after the diagnosis is made.[3] Recently it has become apparent that patients are surviving for longer periods of time and asymptomatic intervals of ten years has already been alluded to.[3] When the slope of the rise of bilirubin is plotted against survival, it is noted that the bilirubin rise is slower in patients now living with the disease as compared to those who have died from it.[3] This may be due to a decreased virulence of the disorder, or to better supportive care now being given to patients. It is hoped that d-penicillamine or other treatments in the future may lead to better survival and perhaps total arrest of the disease.

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Oncology

Improved Survival for Patients with Advanced Non-Small-Cell Carcinoma of the Lung

Thomas P. Miller, M.D.

Editor:
David Alberts, M.D.

Introduction

In 1978, the diagnosis of lung cancer was made in 102,000 Americans, and 92,000 of these are expected to die of the disease. Overall, the five-year survival rate for patients with lung cancer is 8%, which represents only minimal improvement since 1940.[1] These facts indicate a need for better therapy.

Surgery has been the treatment of choice for a minority of patients with lung cancer. At the time of diagnosis, only 20% of lung cancer patients are thought to have resectable disease.[2] The majority of patients with resectable tumors develop recurrent disease within 5 years of surgery. Thus, 90% of all patients diagnosed as having lung cancer die of metastatic disease.

Although the need for systemic therapy in lung cancer is apparent, physicians have been reluctant to treat patients with chemotherapy. In the past it has been difficult to show either palliative or survival benefits for patients treated with chemotherapy. The side effects, toxicities, and costs appeared to far outweigh the objective benefits. However, this appraisal of systemic therapy for lung cancer is no longer accurate. In at least one histologic subtype, small cell carcinoma of the lung, high response rates (>80% in most series) and improved survival (from 7-13 weeks in untreated patients to a median of 1 year) have changed the physician's approach to the patient.[3] Recently, several reports have indicated a survival advantage for patients with extensive non-small-cell varieties of lung cancer. These reports and the experience of the University of Arizona and Tucson Veterans Administration Hospitals are reviewed.

From: Department of Internal Medicine, Section of Hematology and Oncology, University of Arizona Health Sciences Center, 1501 North Campbell Avenue, Tucson, Arizona 85724.

The choice of therapy for carcinoma of the lung depends on the histologic subtype and extent of disease at diagnosis. Histologically, it is essential to recognize the small cell variety, as treatment for that disease is unique. The extent of disease at diagnosis is established using the TNM staging system as advocated by the American Joint Committee for Staging and End-Results Reporting.[4] According to that classification, advanced disease (Stage III) is almost always inoperable. The disease may be inoperable but limited to one hemithorax (Stage III M$_0$) or extensive, with metastases outside one hemithorax (Stage III M$_1$). This distinction has prognostic and therapeutic implications. Patients with Stage III M$_0$ disease survive a median of 5 months, whereas patients with Stage III M$_1$ disease survive a median of 3 months.[5]

Table 1
Objective Response of Non-Small-Cell Carcinoma of the Lung to Single Drugs[6]

Drug	Percent Responding		
	Squamous Cell	Adeno-Carcinoma	Large Cell
Methotrexate	25	24	13
Cyclophosphamide	22	19	20
Mechlorethamine	21	28	26
Adriamycin	14	13	17
Hexamethylmelamine	12	15	18
CCNU	12	12	20
Procarbazine	13	19	35
5-Fluorouracil	14	18	—

Treatment of Extensive Disease

Since most patients with lung cancer have extensive disease at diagnosis or recur with extensive disease after failing local treatment attempts (surgery and radiation), successful systemic therapy must be developed in order to improve survival for this group. A number of drugs are active as single agents in the non-small-cell varieties of lung cancer (Table 1). However, in spite of these response rates, single-drug chemotherapy has not prolonged patient survival (except in small cell carcinoma).[5] Recently, several drug combinations hav been shown to confer a survival advantage o responding patients.[7] [11] Regimens showing improve survival are summarized in Table 2. It is difficult t compare the results of these regimens because o differences in prior treatment, extent of disease, and the grouping of histologic subtypes. However, it is clear that patients who respond to combination chemotherapy (an objective decrease of 50% in the product of two perpendicular measurements of the tumor) survive longer than both non-responders and historic controls. In this respect, all of the studies in Table 2 have shown that non-responders live 2.5-3.0 months, a survival time that is identical to that of historical controls with Stage III M$_1$ disease.[5] Importantly, those patients whose disease stabilized with chemotherapy have attained a median survival nearly identical to that of responding patients. Thus, in every series cited in Table 2, a majority of patients with advanced non-small-cell carcinoma of the lung benefitted from chemotherapy with significantly improved survival.

Table 3
FOMi Treatment Schedule

	Day 1	2	3	4
5-FU, 300 mg/m², iv	X	X	X	X
Vincristine, 2 mg, iv	X			
Mitomycin C, 10 mg/m², iv	X			

This schedule is repeated every 3 weeks for 3 courses, then every 6 weeks.

At the University of Arizona and the Tucson and San Antonio Veterans Administration Hospitals, patients with Stage III M$_1$ disease have been treated with the FOMi combination (Table 3). (5-fluorouracil, vincristine, and mitomycin C). The regimen was extremely well tolerated by the majority of patients. Nausea and vomiting were uncommon side effects. Myelosuppression was common but manageable. There were two drug-related deaths in the group of 56 patients (due to bacterial sepsis). Forty percent of patients

Table 2
Therapy for Advanced Non-Small-Cell Carcinoma of the Lung: Regimens Showing Improved Survival

Regimen*	No. of Responders/No. Treated			Median Survival Responders (Mos)	Reference
	Squamous Cell	Adeno-Carcinoma	Large Cell		
Bleo-COMF	5/14 (36%)	6/17 (35%)	4/7 (57%)	8.5	7
CAMP	9/27 (33%)	8/22 (36%)	—	12.6†	8
MACC	8/22 (36%)	15/29 (58%)	7/20 (35%)	11‡	9
FAM	—	9/25 (36%)	—	8.5+	10
FOMi	—	17/43 (40%)	4/10 (40%)	7.5+	11

†16 patients received radiation to the lung immediately prior to chemotherapy.
‡Includes patients with small-cell carcinoma of the lung.
*Eponyms are defined in the appropriate reference.

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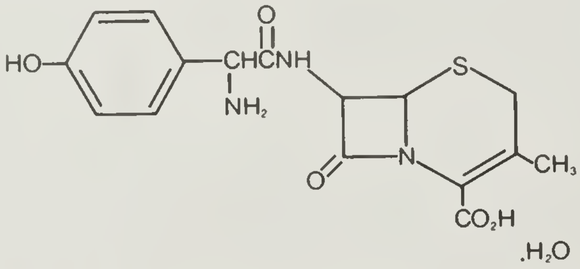
References:
1. Data on file, Mead Johnson Pharmaceutical Division.
2. Gatley MS. To be taken as directed. *J Roy Coll Gen Pract* 16:39, 1968.

DESCRIPTION: DURICEF® (cefadroxil monohydrate) is a semisynthetic cephalosporin antibiotic intended for oral administration. It is a white to yellowish-white crystalline powder. It is soluble in water and it is acid-stable. It is chemically designated as 7-[[D-2-amino-2-(4-hydroxyphenyl)acetyl]amino]-3-methyl-8-oxo-5-thia-1-azabicyclo [4.2.0]oct-2-ene-2-carboxylic acid monohydrate. It has the following structural formula:



Clinical Pharmacology–DURICEF (cefadroxil monohydrate) is rapidly absorbed after oral administration. Following single doses of 500 and 1000 mg., average peak serum concentrations were approximately 16 and 28 mcg./ml., respectively. Measurable levels were present 12 hours after administration. Over 90 percent of the drug is excreted unchanged in the urine within eight hours. Peak urine concentrations are approximately 1800 mcg./ml. during the period following a single 500 mg. oral dose. Increases in dosage generally produce a proportionate increase in DURICEF urinary concentration. The urine antibiotic concentration, following a 1 gm. dose, was maintained well above the MIC of susceptible urinary pathogens for 20 to 22 hours.

MICROBIOLOGY: *In vitro* tests demonstrate that the cephalosporins are bactericidal because of their inhibition of cell-wall synthesis. DURICEF is active against the following organisms *in vitro:*

- *Beta-hemolytic streptococci*
- *Staphylococci,* including coagulase-positive, coagulase-negative, and penicillinase-producing strains
- *Streptococcus (Diplococcus) pneumoniae*
- *Escherichia coli*
- *Proteus mirabilis*
- *Klebsiella* species

Note–Most strains of *Enterococci (Streptococcus faecalis* and *S. faecium)* are resistant to DURICEF. It is not active against most strains of *enterobacter* species, *P. morganii,* and *P. vulgaris.* It has no activity against *Pseudomonas* or *Herella* species.

Disc Susceptibility Tests–Quantitative methods that require measurement of zone diameters give the most precise estimates of antibiotic susceptibility. One recommended procedure (CFR Section 460.1) uses cephalosporin class disc for testing susceptibility; interpretations correlate zone diameters of the disc test with MIC values for DURICEF. With this procedure, a report from the laboratory of "resistant" indicates that the infecting organism is not likely to respond to therapy. A report of "intermediate susceptibility" suggests that the organism would be susceptible if the infection is confined to the urinary tract, as DURICEF produces high antibiotic levels in the urine.

INDICATIONS: DURICEF (cefadroxil monohydrate) is indicated for the treatment of the following infections when caused by susceptible strains of the designated microorganisms:

- Urinary tract infections caused by *E. coli, P. mirabilis,* and *Klebsiella* species
- Skin and skin structure infections caused by staphylococci and/or streptococci

Note–Culture and susceptibility tests should be initiated prior to and during therapy. Renal function studies should be performed when indicated.

CONTRAINDICATION: DURICEF (cefadroxil monohydrate) is contraindicated in patients with known allergy to the cephalosporin group of antibiotics.

WARNING: IN PENICILLIN-ALLERGIC PATIENTS, CEPHALOSPORIN ANTIBIOTICS SHOULD BE USED WITH GREAT CAUTION. THERE IS CLINICAL AND LABORATORY EVIDENCE OF PARTIAL CROSS-ALLERGENICITY OF THE PENICILLINS AND THE CEPHALOSPORINS, AND THERE ARE INSTANCES OF PATIENTS WHO HAVE HAD REACTIONS TO BOTH DRUGS (INCLUDING FATAL ANAPHYLAXIS AFTER PARENTERAL USE.)

Any patient who has demonstrated a history of some form of allergy, particularly to drugs, should receive antibiotics cautiously and then only when absolutely necessary. No exception should be made with regard to DURICEF (cefadroxil monohydrate).

PRECAUTIONS: Patients should be followed carefully so that any side-effects or unusual manifestations of drug idiosyncrasy may be detected. If a hypersensitivity reaction occurs, the drug should be discontinued and the patient treated with the usual agents (e.g., epinephrine or other pressor amines, antihistamines or corticosteroids).

DURICEF (cefadroxil monohydrate) should be used with caution in the presence of markedly impaired renal function (creatinine clearance rate of less than 50 ml/min/1.73M²). (See Dosage and Administration.) In patients with known or suspected renal impairment, careful clinical observation and appropriate laboratory studies should be made prior to and during therapy.

Prolonged use of DURICEF may result in the overgrowth of nonsusceptible organisms. Careful observation of the patient is essential. If superinfection occurs during therapy, appropriate measures should be taken.

Positive direct Coombs tests have been reported during treatment with the cephalosporin antibiotics. In hematologic studies or in transfusion cross-matching procedures when antiglobulin tests are performed on the minor side or in Coombs testing of newborns whose mothers have received cephalosporin antibiotics before parturition, it should be recognized that a positive Coombs test may be due to the drug.

USAGE IN PREGNANCY: Although no teratogenic or anti-fertility effects were seen in reproductive studies in mice and rats receiving dosages greater than the normal human dose, the safety of this drug for use in human pregnancy has not been established. The benefits of the drug in pregnant women should be weighed against a possible risk to the fetus.

ADVERSE REACTIONS: Gastrointestinal–The most frequent side-effect has been nausea. It was infrequently severe enough to warrant cessation of therapy. Administration with food decreases nausea and does not decrease absorption. Diarrhea and dysuria have also occurred.

Hypersensitivity–Allergies (in the form of rash, urticaria, and angioedema) have been observed. These reactions usually subsided upon discontinuation of the drug.

Other reactions have included genital pruritus, genital moniliasis, vaginitis, and moderate transient neutropenia.

DOSAGE AND ADMINISTRATION: DURICEF (cefadroxil monohydrate) is acid stable and may be administered orally without regard to meals. Administration with food may be helpful in diminishing potential gastrointestinal complaints occasionally associated with oral cephalosporin therapy.

Adults–For urinary tract infections the usual adult dosage is one gm. (two 500 mg. capsules) two times per day. For skin and skin structure infections the usual dose is 500 mg. two times per day or 1 gm. once a day.

In patients with renal impairment, the dosage of cefadroxil should be adjusted according to creatinine clearance rates to prevent drug accumulation. The following schedule is suggested. In adults, the initial dose is 1 gm. of DURICEF (cefadroxil monohydrate) and the maintenance dose (based on the creatinine clearance rate [ml/min/1.73M²]) is 500 mg. at the time intervals listed below.

Creatinine Clearances	Dosage Interval
0-10 ml/min	36 hours
10-25 ml/min	24 hours
25-50 ml/min	12 hours

Patients with creatinine clearance rates over 50 ml/min may be treated as if they were patients having normal renal function.

Children–Dosage and safety have not yet been established in children.

HOW SUPPLIED: DURICEF® (cefadroxil monohydrate) capsules 500 mg. for oral administration in an opaque maroon cap and opaque white body No. 0 hard gelatin capsule. On each half capsule printed in black is "MJ" and "500." Available in bottles of 24 capsules (NDC 0087-0784-41) and 100 capsules (NDC 0087-0784-42).

U.S. Patent Re. 29,164

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Mead Johnson & Company
Evansville, Indiana 47721

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eceiving FOMi had an objective response (Table 2). Another 20% of patients had stabilization of disease with FOMi. Both groups, or 60% of all patients treated with FOMi, survived significantly longer than patients who had progressive disease ($p=0.002$). The entire FOMi reatment group (56 patients) survived significantly onger than a group of placebo-treated historical ontrols ($p=0.028$).

Our current approach to patients with extensive non-mall-cell carcinoma of the lung is with combination hemotherapy similar to the FOMi regimen. A new vinca alkaloid, vindesine, has been substituted for vincristine. It is hoped that this substitution will further improve the response rate and response duration.

Treatment of Limited Disease

Patients frequently have disease limited to one hemithorax that is unresectable because of tumor size (Stage III M_0, mediastinal node involvement (Stage II N_2), or medical contraindications to surgery (eg, chronic obstructive pulmonary disease.) Treatment for these patients is also changing.

Traditionally, radiation therapy was reserved for palliation of symptomatic lesions (pain or obstruction). More recently, some centers have taken a more aggressive approach to patients with limited disease by treating all known disease with 5000-6000 rads.[12] In view of some success with higher-dose irradiation and our observed responses to combination chemotherapy in patients with extensive disease, the University of Arizona and Tucson Veterans Administration Hospitals have begun a pilot study for patients with inoperable but limited disease. The combined approach consists of two courses of FOMi, high-dose irradiation of all known disease, and subsequent resumption of chemotherapy with FOMi for an additional six courses.

There is a precedent for such an aggressive approach. Bitran has treated patients with limited disease with 3000 rads of radiation followed by CAMP (Table 2).[13] That pilot study demonstrated improved survival for the entire group (median survival, 9.6 months) and greater benefit for 20/39 patients who responded to therapy (median survival, 15.2 months). These more aggressive approaches to patients with limited disease will have to be confirmed with studies of large numbers of patients. However, it is expected that patients with smaller tumor burdens (limited disease) and better performance status will respond at a higher rate to the combined approach.

Summary

New combinations of currently available drugs have shown modest but significant benefit for patients with extensive non-small-cell carcinoma of the lung. Survival is improved for both patients who respond and patients whose disease stablizes with chemotherapy. Because of the encouraging results in patients with extensive disease, several studies are underway to test the efficacy of combined treatment (radiation and chemotherapy) in patients with limited but unresectable lung cancer.

Chemotherapy for the non-small-cell varieties of bronchogenic carcinoma of the lung has recently improved to the point of practical utility. Physicians should strongly consider a trial of chemotherapy for patients with advanced non-small-cell carcinoma of the lung.

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Peripheral Neuropathy Secondary to cis-Dichlorodiammino-Platinum (II) (Platinol)

Treatment for advanced ovarian cancer

A. C. Marin, M.D.
Bernice Rierson, LPN

Summary

Two cases of partially reversible peripheral neuropathy which occurred during treatment with cis-Platinum are presented. In the first case, a patient with a poorly differentiated ovarian adenocarcinoma developed neurotoxicity after six courses of DDP. In the second case, a patient with an advanced mixed muellerian tumor of the ovary developed similar toxicity after four courses with DDP. Both patients received combination chemotherapy with Hexamethylmelamine (HMM) and Megace. Peripheral neuropathy and nephrotoxicity appear to be the major dose-limiting factors of this compound.

Introduction

Cis-Dichlorodiamminoplatinum (II) (Platinol) until recently an investigational chemotherapeutic agent, is being widely used in patients with a variety of tumors. The major toxicities of this agent have been renal, gastrointestinal, otologic, and hematologic.[1] Less frequently peripheral neuropathy has also been reported.[2, 4] We have observed two more cases of neurotoxicity which developed during therapy with cis-DDP.

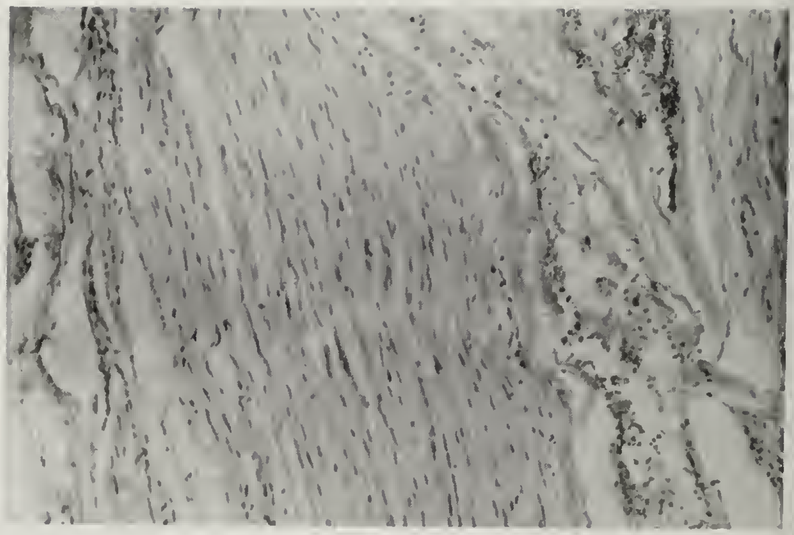

Figure 1
Biopsy of sural nerve H and E. 10X showing areas of degeneration.

Patient One

A 50 year old female underwent an exploratory laparotomy in February 1978 for a pelvic mass and ascites. A widely disseminated ovarian adenocarcinoma was found involving omentum, intestines, liver and peritoneum. A bilateral salpingoophorectomy and biopsies of omentum were performed. Large amounts of tumor including a 3 cms thick omental cake were not resected. A physical examination on March 1978 revealed a large abdominal mass measuring approximately 8 x 8 cms just above the remaining uterus. Neurological examination at this time was unremarkable. A CBC, SMA-12, BUN and Creatinine, revealed no abnormalities. In March 1978 she began to receive cis-Platinum therapy at a dose of 50 mg/m^2 IV push along with Megace 40 mg p.o. TID and Hexamethylmelamine 6 mg/Kg/day p.o. in divided doses. Platinum courses were repeated every three weeks. The patient was hydrated prior to DDP administration.

A complete tumor response was evident after three treatment courses. This was documented by physical examination and ultrasound which failed to demonstrate any pelvic or abdominal masses or reaccumulation of ascitic fluid. Ten days after her sixth course of DDP, the patient noticed the onset of loss of strength, "tight skin" around the hands and wrists, numbness of both legs and difficult ambulation. A neurological examination revealed loss of vibratory and position sense in both lower extremities, loss of vibratory sensation in the hands, loss of position sense in the left hand, a 4x positive Romberg examination and absence of all deep tendon reflexes. Electromyogram showed evidence of polyradiculopathy and a biopsy of the sural nerve revealed areas of myelin degeneration (Figure 1). The patient was placed on high dose Thiamine, and maintenance doses of Hexamethylmelamine and Megace. She has remained in remission 13 months after her first course of chemotherapy and with minimal neurological deficit characterized by reduced deep tendon reflexes, absent vibratory sensation in the legs and shaky handwriting. The patient did have return of position sensation in the left hand and legs.

From: Arizona Health Plan, Division of GYN/Oncology, 925 E. McDowell Road, Phoenix, AZ 85006. Reprint requests to Dr. Marin.

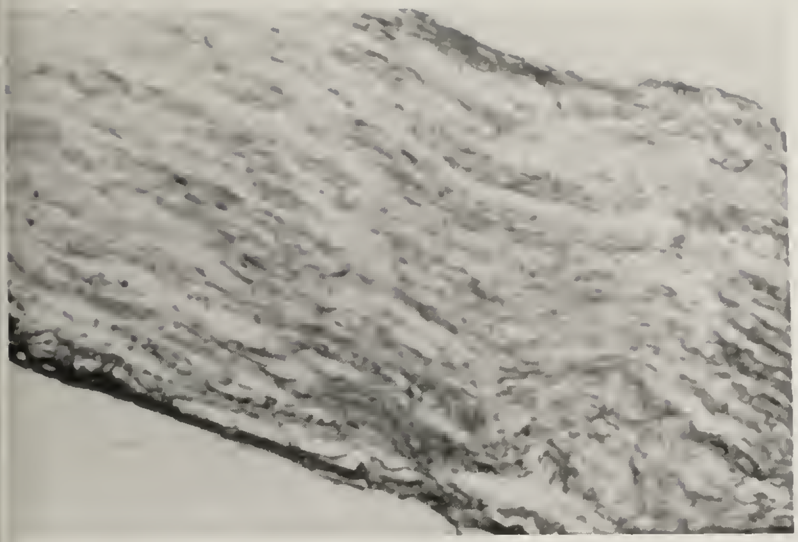

Figure 2

Biopsy of sural nerve Bodian stain. 10X with areas of degeneration of axons and myelin.

Patient 2:

In April 1978 a 56 year old post-menopausal patient underwent a supracervical hysterectomy, bilateral salpingoophorectomy, small bowel resection, omentectomy, appendectomy and tumor debulking for a mixed muellerian tumor of the ovary stage IV. Minimal disease was left after surgery, measuring less the 2 cms in diameter and adherent in the cul-de-sac. In May 1978 after a physical examination which revealed no evidence of persistent tumor and a normal neurological examination, the patient was placed on cis-DDP 50 mg/m^2 IV push every three weeks, Hexamethylmelamine 6 mg/Kg/day p.o. in divided doses and Megace 40 mg p.o. TID.

In September 1978 after four courses of cis-DDP, the patient developed numbness and loss of strength in lower extremities. Neurological examination revealed absence of all deep tendon reflexes, mild motor dysfunction and marked large fiber dysfunction, especially in the lower exremities and in the distal upper extremities.

Electromyogram showed evidence of polyradiculopathy, and a sural nerve biopsy was compatible with myelin degeneration (Figure 2). The patient was started on a high dose Thiamine and maintenance doses of HMM and Megace. She has remained free of disease 15 months after tumor resection and with a presistent motor dysfunction in both legs.

Discussion

Cis-Diamminodichloroplatinum II (Platinol) is a complex formed by a central atom of platinum surrounded by chlorine and ammonia atoms in the cis position in the horizontal plane. The chemical structure is reminiscent of the mustard-type bifunctional alkylating agents. Biological effects include antimicrobial, immunosuppressive and antineoplastic characteristics.[1]

This compound has shown significant activity in several experimental tumors, including B16 melanoma, Walker 256 carcinosarcoma and leukemia L 1210.[3] Pharmacokinetic studies in man have described the distribution and excretion of radioactive DDP after a simple intravenous administration of the drug, suggesting poor penetration into the central nervous system.[4] Pharmacokinetic studies of DDP following a six hour infusion suggest plasma accumulation 3-4 days after injection. In one patient 2.5% of serum levels appeared in cerebrospinal fluid during the first dose and 5% during the second dose, suggesting accumulation which can result in neurotoxicity.[5]

Neurotoxicity has been reported to occur in 21 patients treated with cis-Platinum in phase II studies[4] and was characterized by peripheral neuropathies in both upper and lower extremities. These neurological symptoms generally occurred in patients who received DDP at doses of 2.5 mg/Kg and 15 mg/m^2 daily X 5 and 3-4 weeks intervals with total doses ranging from 500-1470 mg.

A recent report by the National Cancer Institute,[6] has shown that cis-DDP administration can induce inappropriate renal magnesium wasting secondary to a renal tubular defect in magnesium conservation, this can result in serious clinical syndromes of magnesium deficiency manifested by muscular irritability, tremors and a positive Chvostek sign. Hexamethylmelamine (HMM) is a substitute melamine derived from cyanuric chloride. Currently it is an investigational drug sponsored by the National Cancer Institute. The cummulative response for HMM used as a single agent in ovarian cancer is 39%. This degree of activity is in the same range as the alkylating agents.[7]

Among the major toxic effects, gastrointestinal, hematologic and neurotoxicity have been described as dose-limiting factors. However, neurologic toxicity including ataxia is dose related and does not usually include peripheral neuropathy, these symptoms are generally reversible when the treatment is stopped.[8]

Because of the complete resolution of measurable disease in patient l, the unusual reponse in patient 2, and the marked neurological improvement after DDP was discontinued, patients have been placed on daily HMM maintenance doses with no serious adverse effects.

Acknowledgements

We are grateful to Dr. A. J. Castillo for his excellent technical assistance.

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Psychiatric Disorders

Psychiatric Hospital Treatment of the Adolescent

Howard S. Gray, M.D.

Psychiatric hospitals in all countries experienced an influx of adolescent patients during the 1960's. This process is now being evaluated and divergent views regarding the hospitalization of adolescents are emerging.

Studies support that no one hospital-based treatment approach is more efficacious than another and the purpose of this monograph is to set forth those therapeutic modalities and approaches common to the hospital management of all adolescents.

Protection:

Protection is aimed at decreasing or stopping the harmful effects of the patient's environment and/or his behavior. It is incumbent upon the hospital staff to take an authoritarian stance which allows treatment to take place, provide a moratorium on conflicts between the patient and his pre-hospital environment and to initiate early thrusts for mature strivings. Naturally, this phase of hospitalization raises certain ethical issues.

Controls:

These are designed to decrease pathological behavior such as assaultiveness, suicidal attempts, runaways and concomitant physical illness. Needed support is offered and reality testing is enhanced. Acting-out behavior is blocked and the patient is encouraged to verbalize internal conflicts instead. Therapists and hospital staff need to verbally confront the patient and traditional ways of establishing controls, such as restraint and locked doors, may be necessary. The use of the latter techniques may be demoralizing to staff charged with implementing them and the patients's normal expression of feelings may be suppressed if controls are used inappropriately or excessively.

Support:

Supportive efforts aim to make the individual patient subjectively feel better. This modaliaty requires the hospital staff and therapist to get beyond surface behavior to the level of underlying thoughts and feelings. This is accomplished by giving patients individualized attention, Support is particularly helpful during the early phase of hospitalization when new patients are especially vulnerable and when they may often exhibit false bravado. Setting limits, but not confronting too aggressively, is seen by the troubled adolescent as supportive and helpful. Patients with paranoid ideation and chronic character problems such as sociopathy are especially difficult to treat during this phase of hospital treatment.

Structure:

It is necessary for a hospital-based adolescent treatment program to be predictable, consistent and dependable. This is particularly helpful for schizophrenic teenagers who require structure, reliable external reality and continuity. The difficulties

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From: Adolescent Treatment Program, Camelback Hospital, 3352 East Camelback Road, Phoenix, AZ 85018. (Dr. Gray, Assistant Medical Director and Director, Adolescent Treatment Program) encountered in imposing structure include suppression, the possibility of masking pathology and limiting creativity.

Behavior Modification:

This implies the use of the hospital staffs' behavior to encourage and modify the patient's inappropriate and self-defeating behavior. Identification with how staff handles feelings and stress is a crucial therapeutic modality. Modification of behavior is enhanced by built-in systems of rewards and privileges the patient progressively may earn in a level system.

Use of Interpersonal Relationships:

Hospital personnel utilize relationships with patients to improve their social skills, confidence and ability to verbalize problems. During this phase of hospitalization the staff needs to give up their authoritarian role and become more tolerant. The use of the hospital peer group is essential and particularly helpful for passive patients. Various levels of interpersonal relationships include patient-patient interactions, staff-patient interactions and group interactions with other hospital departments. Interestingly, most patients view patient-patient interactions, as carried out in group psychotherapy, as most helpful.

Modeling:

Modeling is similar to behavior modification in that the patient sees how the staff behaves and endeavors to develop similar coping mechanisms. The use of modeling is more important in long-term hospitalization and appears to be more readily accomplished by the employment of younger personnel.

Individualizing:

Here the emphasis is on clarity of what each patient thinks and feels. It is important for the staff to communicate to the patient that it is alright to fail at times and that experimentation and resulting learning is important. Individualizing requires a higher level of functioning on the part of the patient and requires, too, that the staff let go and merely point out to the patient areas of difficulty. Staff needs to become increasingly tolerant and empathic.

Skill Development

Virtually all hosptial departments are involved in aiding the adolescent to develop and refine skills. Educational, vocational and recreational modalities are employed in the context of working with others. Team sports are especially helpful.

The hospital based treatment of the psychiatrically disturbed adolescent is a challenging, often frustrating but usually ultimately rewarding experience for the physician. It may be that treatment teams are not able to accomplish all these modalities equally well, but they represent goals which psychiatrists working with hospital treatment teams should strive to attain.

Acknowledgement

The author gratefully acknowledges his discussions with Doyle Carson, M.D., Medical Director Timberlawn Hospital, Dallas, Texas

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Special Article

Consumer and Physician Attitude Study: An Overview

Stephen W. Brown

Dr. Brown is a Professor of Marketing at Arizona State University. As an expert in consumer research and professional services, he recently conducted a major study for ArMA from which a series of five articles for *Arizona Medicine* have been written. This first article in the series highlights the procedures and results from the complete study.

In recent years the media has put the word "malpractice" into the daily awareness of virtually all Americans. The sue syndrome had compelled physicians to take out malpractice insurance and forced insurance companies to multiply their premiums. According to one respected publication, many physicians have been forced to think of their patients as "potential lawsuits" rather than real people with medical problems.[1]

Physicians, both individually and through medical societies, have been rightfully disturbed by the increased costs of insurance and its unavailability in some cases. Legislators, government agencies, and insurance companies have confronted the problem with hearings, task forces, and in some states, regulatory changes. About the only group unrepresented in these discussions has been consumers. Yet, they are the people who ultimatley pay the bills for escalating premiums, additional "precautionary" tests, and other outgrowths of the malpractice problem. Furthermore, the consumer or patient initiates the malpractice suit based on real or perceived dissatisfaction with the physician.

Various sources indicate that the sue-syndrome among consumers stems, in part, from poor physician-patient rapport. Yet, we know little about the consumers' attitudes toward physicians. Given this scenario, a study was initiated by the Ad Hoc Malpractice Insurance Crisis Committee of the Arizona Medical Association. The overall objective of the study was to examine the opinions, attitudes, and behavior of consumers toward health care and physician services, especially as they may impact on malpractice suits. A secondary objective was to examine physicians' opinions and attitudes on the same topic.

In the preliminary work associated with the study, selected state and local medical associations throughout the country were contacted to see if any similar studies had already been conducted by them. With the exception of a study sponsored by the Ohio State Medical Association in 1976, no similar research was uncovered.[2]

Yet, unsolicited comments from many of the associations suggest that the relationship between the patient and the physician is the key to the malpractice problem. Table 1 highlights some of the observations from selected medical associations.

This article is the first of a series of five that will appear in consecutive issues of *Arizona Medicine*.[3] As the first article, this piece overviews the study's procedures and findings from both the consumer and physician surveys. Succeeding articles will delve into greater detail on the results from both surveys. The concluding article in the series will offer a comparative analysis of the responses from the consumer and physician surveys.

Methods and Procedures

As noted earlier, two surveys were conducted as a part of this study—a consuemr survey and a physician survey. The consumer survey was conducted via a telephone questionnaire adminstered to a random sample of 1,000 adult residents of Arizona during May and June of 1979. More specifically, 546 respondents were sampled from metropolitan Phoenix and 251 from metropolitan Tucson. The remaining 200 respondents came from various communities outside the state's two population centers.

A sample of 1,000 adults is statistically reliable and offers a good base for making inferences about the Arizona adult population. The questionnaire was administered by a professional interviewing firm, M.R. West Marketing Research, through facilities in Phoenix and Tucson, using skilled training and regular monitoring of the interviews. Persons who were not at home were called back to attempt to secure a representative sample. The sample itself was drawn by Western Computing, Inc. of Phoenix.

The physician survey was administerd in May via the mail to a sample of 1,000. The sample was randomly generated by the Arizona Medical Association from the membership roster. 591 physicians responded to the survey. This response rate is more than adequate to make inferences about Arizona physicians as a whole.

The questionnaires for the two surveys were developed through the assistance of several sources, including the responses to the preliminary survey, existing studies on health care and physician services, the judgements of the Association's Ad Hoc Malpractice Insurance Crisis Committee, staff, public realtions consultant, and the expertise of the author of this article. Prior to finalization, both instruments were pretested to make sure that the questionnaires communicated effectively and were generating the intended information. The physician survey contained some of the same questions asked consumers. Accompanying the physician questionnaire was a cover letter from ArMA President William E. Crisp, M.D.

The results from both surveys were professionally coded and then delivered to Western Computing, Inc. for computer analysis. The results coupled with other information provided the inputs for the writing of the overall report and the series of articles for *Arizona Medicine*.

Findings

This section is divided into two parts. The first part overviews the results of the consumer survey. This is follwed by a summary of the findings from the physician survey. Future articles will offer a more detailed account of the results from both surveys.

Consumer Survey. A profile of the respondents to the consumer survey indicates that the sample represents a cross section of Arizona's adult population. All age groups, family sizes, educational levels, income levels, geographical areas and sexes were included in the survey. The fact that almost a quarter of the respondents did not have a family doctor is a relfection of the state's mobile population. In addition to the continuing influx of newcomers, existing residents have a relatively high propensity to move from one place to another within their community of residence. These two factors contribute to the high number of people without a family doctor.

The survey reveals that the bulk of Arizona consumers have a primary physician engaged in either solo practice or practice with others. Various health plans account for just over ten percent of the primary care. Physicians engaged i family/general practice account for near three-quarters of the consumers' prima physicians. The study results show that good number of patients are not loyal t their primary physician. For exampl although nearly three quarters of th consumers had lived in Arizona for ove six years, only 42% had been with th same primary physician for over si years. If one looked at only long tim residents, the figures are even mor striking.

Within the various household interviewed, a variety of physicians ha been seen in the past tweleve months. I 60% of the cases at least one famil member had seen a family/genera practitioner. A third of the household had seen an OB/Gynecologist, a quarte had seen a surgeon, and approximatel 20% had seen a pediatrician and/o internists. Osteopaths had been visite by 10% of the families. The frequency o visits to physicians varied widely.

As part of the survey, consumers wer asked their opinions of physicians i general, their own physician, and patien health interests. From reviewing th responses one can reasonably conclud that Arizona consumers have a essentially positive image of physicians This favorable opinion, however, shoul not lull physicians into a state of apathy The observations show areas of concer and implicitly suggest programs fo improvement.

A clear majority of consumers feel tha doctors are ethical and responsible peopl and competent practitioners, but charg to much for the services they provide. I specifically assessing their own physician a substantial majority indicated they ha a lot of confidence in their doctor and tha their doctor does a good job of explaining medical problems. Despite their praise o their own physician, just over half of the consumers felt that doctors in general d a poor job of explaining medical problems to patients.

Consumers are unclear as to whether doctors are at fault or not in most malpractice suits. These results suggest a certain sophistication among the public. Apparently, a sizeable number do not assume that a physician is in the wrong just because a malpractice suit has been initiated.

Most Arizona consumers display an interest in learning about the topic of health. A substantial majority often read newspaper and magazine articles about health topcis and watch TV programs that discuss health. Vast numbers also are interested in receiving printed information from their doctor explaining their medical problem and treatment. The availability of printed material could help overcome some of the apparent communication problems between physicians and patients.

The survey also took a specific look at the public's attitude toward the malpractice situation. The heavy publicity surrounding malpractice has resulted in four out of five consumers being familiar with the general nature of malpractice insurance. Respondents were asked what they thought caused the cost of malpractice insurance to be so high. This question was asked in an open-ended manner, allowing participants to respond with any thoughts that came to mind. Parethetically, those unfamiliar with malpractice insurance were told what the term means before answering this question. Close to half of the respondents associated high malpractice insurance costs with the publicity surrounding the number and dollar amounts of suits being filed. Patient greed was noted by a third of the participants, while only 20 percent attribute the high costs to doctors being a fault.

Following the question on malpractice insurance costs, the respondents were explicitly asked, "Why do you think patients sue doctors?" The results indicate that the public clearly preceives the patient and the doctor as being the key factors in decisions to bring a suit. Half of the repondents mentioned patient greed as being the cause of suits and just under half noted the mistakes of doctors. Lawyer greed, the judicial system and other factors associated with malpractice suits were rarely mentioned.

Later in the survey, respondents were asked to put themselves into two hypothetical situations. They were first told "suppose that you developed a serious medical problem in which you believed your doctor might be at fault." Given this scenario, They were asked the likelihood that they would sue their doctor for malpractice. The largest percentage of people were undecided. Nevertheless, a third said they were "very likely" or "somewhat likely" to initiate action. The second hypothetical situation stated. . ."suppose that your spouse or parent died and you believed their doctor might be at fault." Here again the results were mixed. In this scenario, however, respondents expressed a stronger tendency to sue. The results from the two hypothetical situations show that a substantial segment of the population would consider initiating proceedings against a physician if they felt they had been seriously wronged. In article three in this series, the results of the sue scenarios will be related to the characteristics of the respondents providing the reader with a profile of the kinds of patients most likely to initiate a suit.

Eight of the nearly 1,000 survey participants had actually brought a malpractice suit. An additional 13 had gone as far as consulting a lawyer, while 55 said that they had seriously considered bringing suit. Among these 76 individuals, personal and doctor influences accounted for the bulk of the reasons for bringing suit. In a future article, a profile of these consumers is presented.

Various proposals have been offered for easing the malpractice problem. Participants were presented with five of these proposals. A clear concensus of support exists for the following:

Greater time spent by your doctor in explaining the potential problems of your medicine or operation.

A law limiting the percentage of settlement that lawyers could receive.

A law requiring that a malpractice claim be reviewed by a panel of expert doctors and lawyers before going to court.

Physician Survey: As noted earlier, a survey of physicians was also conducted as part of the overall study. Five hundred and ninety-one (591) responses were generated from the 1,000 questionnaires that were distributed via the mail. Various specialties, levels of experience, ages, and geographic locations were represented in the physician sample.

Early in the survey, the respondents were asked their opinions of physicians. They feel their fellow professionals are ethical, responsible and competent. A substantial majority disagree with the statement, "most physicians are more interested in making money than the well-being of their patients." In addition, three-fourths agreed that physicians are *not* at fault in most malpractice suits.

Despite these favorable impressions, some negative responses were generated. Over half of the participants feel that doctors do a poor job of explaining medical problems to patients. To help physician-patient communications, some experts advocate the development and distribution of brochures for patients. Over half of the respondents were favorable toward this idea.

The physicians were also queried about their preception of how the public views physicians and health care. The vast majority of the participants believe that the public:

—has a lot of confidence in physicians.
—often reads newspaper and magazine articles about health topcis.
—feels that most physicians are ethical and responsible people.
—often watches TV programs that discuss health.
—feels that physicians do a poor job of explaining medical problems to patients.
—feels that physicians charge too much for the services they provide.

Just over half believe that the public feels physicians place money interests at a higher priority than patient well-being. Nearly three-fourths of the respondents also feel that patients would like to have brochures available from them explaining medical topics. The greatest physician disagreement arose over whether the public feels that physicians are a fault in most malpractice suits. The results suggest a high level of uncertainty among physicians as to what the public perceives about doctors and malpractice suits.

Table 1
Observations of Selected Medical Associations on the Relationship Between Patient-Physician Rapport and Malpractice

Medical Association	Comments*
Washington State	"We suspect that lack of understanding on the part of the patient, and poor commmunication between the physician and the patient, contributes to the number of malpractice suits when the outcome of medical care is not as anticipated."
Kentucky	". . .if a patient does have a good relationship with the physician he or she is much less likely to sue the physician if problem arises."
Indiana	"It is a well known fact that the doctor's attitude toward his patient has a great impact on a patient's decision to file a suit."
North Carolina	"Advised by our legal counsel. . .the rapport between patient and physician has a strong influence on the patient's decision to make a claim against or sue the physician and that good rapport has a great deal of influence on avoiding a professional liability suit."

**These comments were generated from a letter from ArMA to these state medical associations last fall. The letter asked if the state association had ever conducted a study of consumer attitudes toward physician services and medical malpractice. The comments above represent the thoughts of selected Associations on patient-physician rapport and malpractice.*

The respondents were asked about factors contributing to the increase in cost of malpractice insurance and factors leading to malpractice suits. A majority of physicians attribute rising insurance costs to lawyers. Approximately a third of the respondents cited the number and dollar amount of suits being filed and the court system (e.g. judges, juries) as culprits. "Doctors making mistakes" was given as the primary reason that patients sue doctors. Patient greed, along with anger and frustration were also common explanations. Nearly a fourth of the respondents mentioned the encouragement of suits by greedy lawyers.

The physician participants were also asked their opinions of five suggestions to help solve the malpractice problem. The respondents strongly favored the following proposals: A law requiring that all disputes between patients and physicians be submitted to a panel of expert physicians and lawyers for arbitration.

A law limiting the percentage of the settlement that lawyers could receive.

A law requiring that a malpractice claim be reviewed by a panel of expert physicians and lawyers before going to court.

Substantial support also exists for the suggestion of spending more time in explaining to patients the potential problems of a particular medicine or operation.

Conclusion

This article is the first of a series of five that will appear in consecutive months in *Arizona Medicine*. All five articles feature the results from a major study sponsored by ArMA. The study focused on consumer and physician services, medical malpractice, and patients. The catalyst for conducting of the study was the belief that consumer - physician communication and understanding are fundamental to the malpractice program.

As the first article in the series, this presentation highlighted the procedures and results from the complete study. In future articles many of the findings summarized in this article are examined in more detail. The second and third articles will offer greater insight on the findings from the consumer survey. The physician survey responses will be discussed in the fourth piece. The fina article will offer a comparative analysis of the consumer and physician findings Together, the five articles will provide the reader with a comprehensive view of the public's and physicians' opinions on physicians, malpractice, and patients.

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3. The five articles are derived form the report *Consumer and Physician Attitudes Toward Physician Services, Medical Malpractice, and Patients." The report was submitted by this author to the Ad Hoc Malpractice Insurance Crisis Committee in August 1979.*

AM State of the Art

Topical Corticosteroids

Norman Levine, M.D.

Editor:
Eric H. Prosnitz, M.D.

Corticosteroid preparations are synthetic analogues with cortisol-like hormonal action which are widely used in dermatology. During the past 20 years they have dramatically reduced the morbidity of certain cutaneous diseases. Although a great deal remains to be discovered about the actions of steroids, tremendous strides have been made in our understanding of the mechanisms of action, uses, and potential problems of topical corticoids. This paper shall summarize some of these advances and shall also discuss current concepts of topical corticosteroid therapy.

Biochemistry and Pharmacology

The physiological properties of corticosteroids are divisible into two broad categories, mineralocorticoids, those influencing fluid and electrolyte balance and glucocorticoids which influence the metabolism of carbohydrates, fats, and proteins. Although all topical steroids have had the mineralocorticoid effect eliminated, it has been impossible to dissociate the anti-inflammatory actions from the glucocorticoid effects, thus the topical agents are also referred to as "glucocorticoids".[1]

The basic steroid molecule on which all subsequent modifications have been derived is hydrocortisone (Figure 1). The most successful modifications have involved increasing the lipophilicity of the molecule, thus improving its penetration through the stratum corneum. This was initially performed by masking the hydrophilic hydroxyl groups with fluorine atoms (thus the term "fluorinated steroids") and other simple

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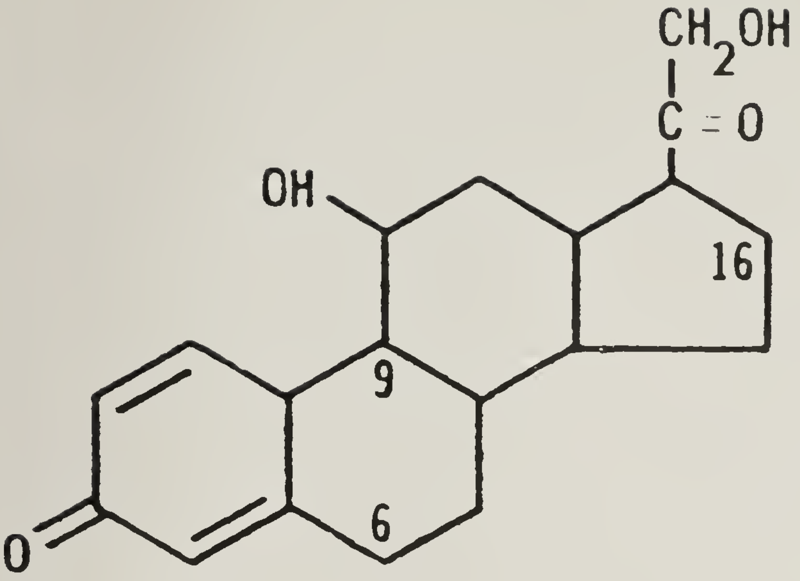
From: Section of Dermatology, University of Arizona Health Sciences Center, Tucson, AZ 85724.



HYDROCORTISONE

constituents. Other minor alterations of the basic molecule not involving fluorination have greatly increased potency so that one can no longer predict the effects (and side effects) of a compound by simply knowing whether a halogen atom is in the structure. In fact, some new, supposedly potent corticosteroids are not halogenated at all.

It is clear that topical glucocorticoids exert a profound anti-inflammatory effect locally but the mechanism of action is not clear. Several competing but not mutually exclusive theories have been proposed to explain the drug action. A lysosomal membrane stabilizing effect may be important.[2] Hydrolytic lysosomal enzymes play an important role in cutaneous inflammation and may be one of the signal events for the inflammatory cascade.[3] No convincing direct in-vivo evidence has been presented in support of this hypothesis but it remains a theoretical possibility.[1]

There is a considerable amount of evidence indicating that topical corticosteroids have an anti-mitotic effect on epithelial cells[4] which is not cell-cycle specific and may be secondary to a reduction of most local macromolecular synthetic activity. One could hypothesize that important inflammatory mediators are not produced so local inflammation does not ensue. The direct anti-mitotic action might also be responsible for the anti-proliferative effect seen in such diseases as psoriasis.

Prostaglandins play an important part in inflammatory processes and some feel that topical steroids act by inhibiting them. In vitro studies do not support this view.[5] Perhaps a cofactor present in skin is necessary for the anti-prostaglandin effect to be seen. This is not the case, however with non-steroidal topical anti-inflammatory drugs such as indomethacin.[5]

Another attractive hypothesis of steroid anti-inflammatory action is the "vascular" theory. It is well known that topical glucocorticoids induce vasoconstriction and the degree of vasoconstriction correlates well with clinical effectiveness.[6-7] In fact, this is one of the criteria on which different compounds are compared. Perhaps circulating mediators of inflammation are blocked from the local site by the vasoconstriction produced by glucocorticoids. Although an attractive hypothesis, there is no evidence to support this concept. It is also possible that the anti-inflammatory and vasoconstrictive properties of corticosteroids act in parallel but are functionally unrelated.

Although the newer, more potent steroid analogues owe most of their increased effectiveness to their inherent potency, clinical efficacy, as well as toxicity are related to the degree of corticosteroid penetration into the skin. The stratum corneum (horny layer) is the only serious barrier to penetration; once a corticoid enters the epidermis, it freely diffuses deeper into the dermis where it may be picked up by the dermal blood vessels and enters the systemic circulation. The stratum corneum is such a formidable barrier, however, less than 1% of hydrocortisone cream applied to the surface will penetrate. Disruption of the stratum corneum is a very effective way of enhancing delivery of corticosteroids. Application of polyethelene film (Saran wrap^{r}) causes increased sweating which hydrates the horny layer which makes it a less effective barrier. Exogenous hydration using wet dressings accomplishes the same purpose. These techniques may increase penetration up to 100 times over control. The skin over certain areas of the body, the scrotum, face, and mucous membranes particularly, are inherently more permeable so high levels of penetration are possible without disrupting the horny layer.

Therapy

To many non-dermatologists, every conceivable dermatosis requires topical corticosteroid therapy. In fact, relatively few skin diseases respond to this mode of treatment. The dermatitis (eczema) group of diseases are the main indications for topical steroids. The common feature of all of these diseases is some degree of epidermal edema (spongiosis) and dermal inflammation. Such diverse underlying processes as atopic dermatitis, seborrheic dermatitis, contact dermatitis, dry skin eczema, and neurodermatitis all present with similar features and all respond to topical corticosteroids. In spite of some controversy over the use of topical steroids in psoriasis,[8] most feel that this disease also responds well to topical glucocorticoids. These agents are used extensively in a number of dermal inflammatory and degenerative processes such as lichen planus, cutaneous lupus erythematous, granuloma annulare, and necrobiosis lipoidica diabeticorum but the responses here are far less certain and often intralesional or systemic corticosteroids are necessary.

The temptation is great to use topical corticosteroids for a number of pruritic disorders as a local "anti-pruritic

Table 1
Potency of Topical Corticosteroids

Group*	Generic Name	Trade Name
I	Betamethasone diproprionate ointment	Diprosone 0.05%
	Halcinonide cream/ointment	Halog 0.1%
	Fluocinolone acetonide acetate cream/ointment	Lidex 0.05%
	Desoximetasone ointment	Topicort 0.25%
	Fluocinolone acetonide acetate gel	Topsyn 0.05%
II	Betamethasone diproprionate cream	Diprosone 0.05%
	Triamcinolone acetonide cream	Aristocort 0.5%
	Betamethasone-17-benzoate gel	Benisone 0.025% Fluorobate 0.025%
	Betamethasone-17-valerate lotion/ointment	Valisone 0.1%
III	Triamcinolone acetonide ointment	Aristocort 0.1% Kenalog 0.1%
	Flurandrenolide ointment	Cordran 0.05%
	Fluocinolone acetonide cream	Synalar HP 0.2%
IV	Triamcinolone acetonide cream/lotion	Kenalog 0.1% Aristocort 0.1%
	Betamethasone-17-valerate cream	Valisone 0.1%
	Fluocinolone acetonide cream	Synalar 0.025% Fluonid 0.025%
V	Desonide cream	Tridesilon 0.05%
	Flumethasone pivalate	Locorten 0.03%
VI	Hydrocortisone cream/ointment	Hytone 1.0% Cort-Done 1.0% Lubricort 1.0%
	Fluoromethalone cream	Oxylone 0.025%
	Prednisolone cream	Meti-Derm 0.5%
	Methylprednisolone acetate cream	Medrol 1.0%

Adapted from Stoughton, R.B. "Evaluation of new potent steroids: in *Recent advances in dermatopharmacology* Frost, P., et al eds Spectrum, New York, 1978.

agent" but the results are generally unsatisfactory. Examples of this are urticaria, pityriasis rosea, insect bites, and pruritis associated with pregnancy, end-stage renal disease, obstructive hepato-biliary disease and other systemic diseases.

A number of diseases may actually be made worse by topical steroid applications.[9] Acne vulgaris can flare when potent steroids are applied to the face as can rosacea. One can alter the appearance of cutaneous fungal infections using steroids by eliminating the inflammatory component, thus making diagnosis more difficult. This is called "tinea incognito." Some feel that viral infections such as herpes zoster or herpes simplex may be made worse by topical steroids.[9] Topical glucocorticoids may impede healing of cutaneous ulcers when the medication is applied directly into the ulcer cavity such as when treating a stasis dermatitis with a central ulceration.

The number of topical steroid preparations available to the practitioner today is enormous and the conflicting claims of the drug manufacturers makes comparison of the products difficult. Two elements of each preparation should be considered, the active steroid molecule and the vehicle in which that molecule is delivered. The inherent potency of the steroid molecules have been extensively evaluated using various minor modifications of the McKenzie-Stoughton vasoconstrictor assay, since the degree of vasoconstriction and potency are closely related.[10] By this assay, six fairly distinct groups can be determined (Table 1), the agents in group I being the most potent and those in group VI having the least effectiveness.

Using the same steroid molecule but altering the vehicle can change the potency somewhat. In general, ointment bases are more potent that creams which in turn are more effective than lotions.[11] Gel formulations which are semisolid emulsions which liquify on contact with the skin have a variable potency depending upon the nature of the gel and the steroid. Ointments owe their increased effectiveness to the occlusive property of the greasy vehicle. Lotions are least occlusive and thus least potent but are the most cosmetically acceptable.

In choosing the appropriate vehicle, the nature and location of the dermatosis as well as the aesthetics of the preparations should be considered. For dry, scaly lesions in non-intertriginous areas (thus not subject to maceration) ointments are often preferred. For large coverage areas where a greasy vehicle would be messy and too occlusive, a cream vehicle is ideal. This is often slightly drying so is useful in "moist lesions." For hair-bearing areas such as the scalp, lotions or gels are more acceptable bases.

There are a number of factors which determine which strength of steroid is indicated. In all patients, the least potent preparation capable of clearing the eruption is always preferred. For lesions on the face, intertriginous areas or on the scrotum, a group VI drug (preferably hydrocortisone) should be used first, particularly if one anticipates greater than two weeks of treatment. For eruptions involving the hands or feet where penetration of the drug through a thick horny layer is a problem, group I drugs should be used. For most other eruptions group IV preparations are appropriate. If the clinician becomes familiar with one preparation from groups I, IV, and VI, he can successfully treat greater than 95% of all steroid responsive dermatoses.

Since the barrier to percutaneous penetration for steroids is so imposing, other techniques are sometimes necessary to achieve a therapeutic result. Two techniques are very helpful in this regard. Polyethelene (Saran wrap[r]) occlusion of the topical steroids or open tap water wet dressings over the steroid should be considered whenever corticosteroids are applied, particularly on the hands and feet. The longer the period of occlusion, the greater the effect but side effects such a folliculitis or miliaria (sweat retention) may interfere so the wrap generally should be applied for no longer than twelve hours per day (or overnight). [12]If the entire body is to be treated, a plastic warmup suit (sweat suit) works well.

If wet dressings are used, 30 minute applications twice a day are usually sufficient. This is especially suitable if the hands alone are being treated since a cotton garden glove can be used repeatedly as the wet dressing vehicle.

With occlusive dressings, the differences between various corticosteroid preparations are diminished so that less potent (and less expensive) agents may be used.[13] There is some evidence to suggest that if occlusive dressings are used the steroid need only be applied one time per day since the glucocorticoid appears to be in a reservoir in the stratum corneum and is somehow released after occlusive dressings are used. In fact, the duration of this effect may last up to 15 days[14] so theoretically, a patient need only apply the corticosteroid every few days as long as repeated occlusive therapy is performed.

As the dermatosis improves one might consider changing the treatment by firstly, discontinuing occlusive dressings and then by changing to a less potent steroid preparation if possible. Many chronic dermatoses such as atopic dermatitis can easily be managed with hydrocortisone alone once initial control is achieved with a more potent agent.[15]

Standard practice has dictated that the more frequent the application of topical glucocorticoids, the more effective the treatment. In recent years this notion has been challenged by a series of experiments which show that with increasing frequency of application, the rate of response as measured by the vasoconstrictor assay[16] and by an assay measuring inhibition of DNA synthesis[17] begins to fall, irrespective of the inherent potency of the steroid used. If one can translate this into clincial effectiveness, perhaps too frequent applications can actually hinder healing. Instructing the patient to apply the corticosteroid preparation twice a day is probably a prudent and effective dosage schedule.

As with many other medications, with increased effectiveness of either the steroid, the vehicle, or the mode of delivery of the steroid, (eg, occlusion), the incidence of side effects becomes increasingly more significant. Patient education about the proper use of the drugs can prevent most of the complications.

Perioral dermatitis, a persistent facial eruption with papules and vesiculopustules of the perioral and perinasal area occurs mainly in women who use the more potent steroid preparations over long periods of time. Unfortunately, the initial perioral eruption is often treated with a still more potent corticosteroid which exasxerbates the process. Attempts at discontinuing the steroids may lead to a rebound dermatitis. The treatment for perioral dermatitis is hydrocortisone cream and a short course of oral tetracycline.[18] A rosacea-like dermatitis as well as a steroid-induced acne of the face may also occur if prolonged use of potent steroids are used on the face.

Cutaneous atrophy involving the epidermis, dermis, and even the subcutaneous tissue occurs particularly after prolonged periods of repeated occlusion of steroids or by repeated application of potent glucocorticoids in body fold areas (axilla, groin, etc.) which provide natural occlusion. In small children occlusion is not necessary to produce the effect. The mechanism of this phenomenon in the dermis may be a combination of inhibition of collagen synthesis by fibroblasts and a stimulation of dermal catabolism of dermal collagen.[19] The lesions present as shiny, depressed transparent areas of skin with telangiectasia, ecchymoses and often striae. Early atrophic changes can be reversed if topical steroid therapy is discontinued.

Through their immunosuppressive and anti-mitotic effects, topical glucocorticoids may adversely modify cutaneous infections. Intraoral steroid applications can induce pseudomembranous candidiasis (thrush).[18] Dermatophyte (cutaneous fungal) infections may be masked by topical steroids by eliminating the inflammatory component, allowing the fungus to flourish. Other infections including scabies and perhaps molluscum contagiosum may be made more extensive with the use of glucocorticoids.

Numerous adverse effects have been observed with the use of steroids applied to the cornea, conjunctiva, and even the eyelids alone (although it is possible that these patients had accidental conjunctival contamination). Increased intraocular pressure with subsequent glaucoma is the most common side effect and has been reported even with very mild steroid preparations if used for a prolonged period.

Table 2
Disease Response to Corticosteroids

Diseases often responsive to topical corticoids	Diseases sometimes responsive to topical corticoids	Diseases rarely responsive to topical corticoids
Atopic dermatitis	Lichen planus	Generalized pruritus
Seborrheic dermatitis	Cutaneous lupus	Pityriasis rosea
Contact dermatitis	Granuloma annulare	Urticaria
Neurodermatitis	Necrobiosis lipoidica	Insect bites
Dry skin (xerotic) eczema	Sunburn	Keloids
Psoriasis		Acne vulgaris

Several miscellaneous side effects of theapy have been reported including increased facial hair after application of steroids in contiguous areas, reversible hypopigmentation and an occasional case of contact dermatitis to the medication itself.

The most important potential side effect of topical corticosteroid therapy is adrenal suppression. Rare cases of iatrogenic Cushing's syndrome, particularly in small children as well as a suppression of the pituitary-adrenal axis with impairment of the normal response to stress have been reported. In adults these effects have been noted almost exclusively in patients using frequent prolonged applications of potent steroids over large surface areas under occlusive dressings, while in small children less therapy has achieved the same effect. It is likely that subclinical defects in pituitary-adrenal axis function occur fairly commonly with treatment of extensive dermatoses. In adults, recovery of function occurs rapidly upon cessation of treatment but in children, this recovery may be somewhat delayed. There is a marked individual variation between patients, however, in their ability to devleop and recover from the adrenal suppressive effects of glucocorticoids.[20] The possibility of adrenal suppression should not cause one to deny a patient the maximum treatment of serious dermatoses but one should tailor therapy so that occlusive dressings and more potent steroid preparations are used only when indicated for a minimum period of time.

Summary

Topical corticosteroids are a group of compounds derived from the hydrocortisone molecule with significant anti-inflammatory and antimitotic activity. The chief use of these drugs is in eczematous eruptions but they are also effective in psoriasis, lichen planus, and perhaps in dermal degenerative diseases and cutaneous lupus. The effectiveness of therapy depends on optimum penetration of the compounds through the horny layer. This is accomplished by hydrating the stratum corneum with occlusive dressings or wet dressings.

There are qualitative differences in steroid preparations and the use of an individual compound should depend on the potency needed to ameliorate the dermatitis as well as the vehicle which delivers the preparation optimally and satisfies the aesthetic needs of the patient.

The side effects of topical steroids are largely preventable and include perioral dermatitis, rosacea-like eruptions, cutaneous atrophy and adrenal suppression.

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Improving Medical School Teaching

The continuing and growing need for medical faculty to become better teachers has its roots in the improving quality of medical students, rapidly advancing information and technology, and growing pressures for social accountability. Each year this College of Medicine has been able to attract better and better faculty members who possess up-to-date knowledge in their specialization, a significant amount of experience in applying their knowledge, and an aptitude for acquiring new information.

> *Yet, "the medical school exists* **primarily** *for the education of the medical students. We (the faculty) can do about everything else we do in another setting, but medical student education requires a medical school. We do have other obligations and prioritites as a faculty, but must meet this one first and best." Vincent A. Fulginiti, M.D., Head, Department of Pediatrics.*

However, due to the very rigor of their preparatory specialty and research activities, new faculty members and their more established colleagues can not be expected to have had a significant amount of formal training on "how to teach".

That which they know about teaching and are able to exercise has often been learned informally from observing their own teachers, teaching assistants, or colleagues. In the absence of appropriate criteria to judge these teaching performances and without specific instruction in learning from such observations, the benefits of such observations are questionable. Some faculty members are left in the position of "getting by" through trial or error. Others manage only through a sincere and deep interest in teaching. The approaches to the development of fundamental teaching skills are individually inefficient and institutionally ineffective. Only a systematic, continuing and College-wide effort can accomplish noticable instruction improvement with the required efficiency.

To insure the effectiveness of such an effort, the following activities are available through the College's Office of Medical Education:

—Provision of personal educational consultation for the observation, diagnosis, and development of specific teaching skills.

—The availability of educational assistance in the development of curricular materials such as course outlines, course objectives, and syllabi.

—Technical assistance in the development of teaching aids and learning materials such as handouts and self-assessment tests.

—Acquisition of and access to readings and references related to teaching skills, instructional research, and learning principles.

—The availability of test design, construction, scoring and analysis services.

—Facilitation of individual or department educational improvement activities or educational inquiries.

—The collection and transmission of student feedback about instruction to assist in identifying specific course and individual opportunities for improvement.

—The dissemination of reliable research results and theoretical contributions which have direct utility.

—The stimulation of interest in effective innovative practices through topical conferences, seminars, or external consultation.

While the approach is just beginning, constant and accumulating instructional impact is expected as these activities become a permanent program staffed by appropritely skilled personnel. The Office of Medical Education is staffed by members who model the personal openness, confidence, sincerity, scholarship, productivity, and teaching skill expected in their faculty clients. An active program of academic inquiry and research together with planned professional development assists with these requirements.

The activities listed are a part of a voluntary educational improvement service provided through central administration. This type of faculty resource is guided by the philosphy that medical faculty members need not be forced or coerced through their vested interests to improve; rather, they are joined by academic professionals who view collaborative instructional improvement as facilitating the productivity of teachers. if a faculty member is persuaded that certain new ways of behaving instructionally will improve his/her performance while maintaining personal security and professional status, he/she will try to adopt these new ways.

As with any social change, individual members of the population served by the change respond with varying speed. The innovators are the easiest adopters of change though the smallest in numbers. They are followed by a sizable majority who have watched and waited to see the proposed practices succeed in actual trial. As priorities and values slowly change, most remaining faculty members are expected to become involved and eventually influenced . Only a small percentage will remain somehow aloof, immune or disinterested. The total process will be slow.

To persist and to succeed, those involved must be dedicated to the development of individual professional teachers who have the ability to produce high quality learning for significant application. This dedication is supported by a deep administrative commitment to educational renewal as evidenced by the provision of social incentives for specific and continuing teaching improvements including personal recognition, teaching awards, promotion in rank and merit compensations.

Robert F. Rubeck, Ph.D.
Acting Director, Office of Medical Education

Louis J. Kettel, Dean
College of Medicine

Auxiliary Highlights

Coconino County

Coconino County Medical Society Auxiliary is excited about the success of its new health project, MATCH—Medical Aid to Children. More than 50% of Flagstaff's school children have already been registered in the program.

Parents fill out and return a form giving consent for emergency medical treatment for their children if the parents cannot be located. Parents are asked to list the childs immunizations, note any allergies, and indicate which physician they want contacted in case of illness or injury. The permission forms are then indexed and filed at the emergency room of the community hospital.

The Auxiliary was asked to coordinate the program, which is funded by a local insurance company and banks. Good publicity through the schools, news radio/TV helped insure its success.

Auxiliary members take turns working one day a week at the hospital, filing consent forms as they come in. Each spring new forms will be sent out, so records can be updated by the time school starts again in September. Looks as if it will be an on-going project for the Auxiliary!

Mrs. Paul Lundstrom, Coconino Auxiliary president, heads the program and is enthusiastic about it. "In the past we've had children sitting in the emergency room for two or three hours—perhaps with a broken arm—because parents couldn't be located to give their consent for medical treatment."

Shape Up for Life

A group of Coconino Auxilians is Shaping Up for Life by hiking up some of the North Country's most picturesque trails. Mrs. Henry Poore is chairman of the interest group, which hikes together twice a month. On one of the hikes they join forces with members of the NAU Faculty Wives Club.

The hikers carry lots of water, lunches and day packs because their jaunts are not just casual little strolls. One of their recent destinations was Mt. Eldon, in the San Francisco mountian group, and that's a steep climb!

When there is snow on the ground in Flagstaff, the ladies plan to hike trails in the Sedona area or go cross-country skiing.

Active Volunteers

Several popular Auxiliary projects are being continued. Coconino plans to take its Nutrition Puppet show to schools in Page, Sedona, Williams and Tuba City. Flagstaff school children saw the show last year.

The traditional Christmas Dinner and bazaar for doctors and their spouses is the Auxiliary's big fund-raising event. Members contribute handmade articles for the bazaar. Proceeds go to AMA-ERF and local charities.

Auxiliary members will again assist the Medical Society with arrangments for its annual Summer Seminar, the last weekend in July. It's a happy combination of medical lectures and the Flagstaff Summer Arts Festival with its concerts and plays. This past year, Auxiliary members sponsored a morning coffee at the home of Dr. and Mrs. Francis Maher and a tour of the Navajo show at the museum of Northern Arizona for wives of attending physicians.

In addition, individual Auxiliary members will continue to give many hours of volunteer service to the Crisis Nursery, Coconino County Guidance Center, Big Sister Program, and the Spouse Abuse Center.

Mrs. John T. Clymer, President
Arizona Medical Association Auxiliary

Correspondence

Editor—

After reviewing the three articles that included my own in October's *Arizona Medicine* (Vol. 35, No. 10, pp 771-776) we can draw some common concerns from dedicated physicians that are shared by many.

1. Physicians are greatly concerned over their loss of income when getting invovled in something new. Most people fear any scheme that threatens their livelihood and physicians are no different than anyone else in that regard.

2. Physicians are greatly concerned over some outside agency such as the government or a large insurance company telling us how to practice medicine. We have been trained to believe that only our world will cause damage to the quality of care.

In my deliberations and discussions all over the valley since the IPA concept has started to be discussed, I have preceived one more unspoken concern that is a larger factor in this process of socioeconomic change.

3. Specialist physicians are becoming very concerned that there may be an excess amount of other doctors in their own specialities and if a system be devised that gives control to those lower cost "not as well trained primary physicians", then the specialists will lose patients and they will have all of their three fears realized.

We have letters at Valleycare from specialists who want to join the IPA if:

1. We guarantee them an exclusive in their specialty.

2. We guarantee them a fixed volume of business.

3. We guarantee that their downside risk will be fixed at a certain level, etc.

We have been told by specialists that most of their referrals come from either other specailists or from other patients within their practice and to some degree they are practicing primary care medicine already.

Another specialist told us that every

new patient coming into his office was charged $65.00 for a first visit, complete history and physical that he can do in fifteen minuts rather than an hour complete history and physicial done by a GP, because specialists are much better trained than generalists and can work fast.

What it boils down to is that specialists really feel that their dollar and patient volume wil fall when the generalists take over and therfore IPA's are not good for medicine!

Our answer to the above is not simple, but represents our view of the reality of the situation.

1. The cost of medical care in the United States is rising at such a rate that the economy can't deal with it and unless the medical profession does something about it themselves, the government is going to place regulatory controls on it and no one, patients or doctors alike will approve of it.

2. The most excessive costs in the system are basically in the hospital setting and far outweigh the cost in the generalists' or specialist's office. The fears of the specialists notwithstanding as described above, the dollar amounts spent in private physician's offices are only a drop in the bucket as compared to the cost realted to hosptial stays and procedures.

3. Valleycare's definition of a primary physician is as follows: Any physician who cares for the entire patient and not just one organ system, and who doesn't self-refer for specialist type procedures without a consult from a primary physician.

Famliy physicians and pediatricians are automatically considered primary doctors. The gray zone area of certain internists and OB/GYN physicians can be considered either way, depending on how they run their practice. According to our Bylaws, a majority vote of the existing primary physicians will settle any questions once an itnernists or OB/GYN declares himself to be in one of the other categories.

4. Valleycare feels that specialists are essential to the medical system and are better than generalists for things that would be handled best by specialists. Our payment system is set up to penalize the primary doctor who doesn't refer the patient who should be referred to a specialist.

The specialist will send in his bills at the usual and customary rates and pending the risks that all providers will be subject to, and will be paid based on those regular rates.

5. No individual specialist will be guaranteed any fixed total amount of dollars or exclusives, but all specialists will be guaranteed that all speciality care patients will go to specialists within the plan.

6. Duplication of work will not be allowed, and if for instance the primary doctor does a complete physical and then refers the patient for a specialist procedure, the specialist will not be allowed to charge for another complete physical primary to doing the procedure (i.e., if the family doctor does a complete physical on an ulcer patient and does an upper G.I. x-ray that indicated need for a gastroscopy, the gastroenterologist will be allowed to charge his full and regular price for the gastroscopy and any routine office followup calls, but will not be allowed to perform another complete exam and charge for it unless it can be medically justified and not just a duplication of existing work.

7. All doctors, generalists and specialists alike, will be given incentives commensurate with their risks to save hospital dollars. As I said in my last article, we all know surgeons who get routine gallbladder patients out of the hospital in four days, while others keep them eight days.

Those extra four days cost close to $1,000 dollars in Phoenix. Those dollars will end up going back to the doctors as a bonus in our plan.

8. As I further alluded to, at one of our meetings, all of us know that keeping a patient in the hospital over a weekend, waiting for Moday morning x-rays can waste two hospital days. That likewise is a $500.00 charge. Those dollars will end up going back to the doctors as a bonus in our plan if a way can be found to get around it.

We see as our major hurdle to fending off government socialized medicine is for doctors to get up the courage to accept that we are what we are.

Each of us knows deep inside, whether or not we are a good doctor and doesn't need to rip anyone off to earn a living. There is true waste of dollars in the medical system as it stands right now and the people that will lose out, when the waste is stopped will not be the patients or the doctors who practice "quality medicine."

If we really believe in the American free enterprise system that has made this country strong, we should not be afraid to set up a medical system with risk and incentive and let the chips fall where they may.

There will always be plenty of sick patients to care for and a good doctor should be the ones left around to care for them.

Copies of our 65 pages of rules and regulations along with applications for membership in Valley Independent Physicians (VIP) are available upon writing to our office at 2525 West Greenway Road, Phoenix, AZ 85023.

Bruce E. Shelton, M.D.
Medical Director

AM
ARMA

President
William E. Crisp, M.D.

President Elect
John T. Clymer, M.D.

Vice President
Clyde W. Kurtz, M.D.

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Executive Director
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Burnell R. Brown, M.D.
Walter V. Edwards, M.D.
C. Herbert Fredell, M.D.
Benjamin K. Harris, M.D.
John W. Kennedy, M.D.
William B. McGrath, M.D.
John Stoner, III, M.D.
William C. Weese, M.D.
Donald J. Ziehm, M.D.

ArMA Reports

THE MINUTES APPEARING IN THIS SECTION HAVE BEEN EDITED TO CONSERVE SPACE. A COMPLETE COPY OF THE MINUTES OF ANY MEETING WILL BE MAILED TO ANY MEMBER REQUESTING THEM.

BOARD OF DIRECTORS

The meeting of the Board of Directors of the Arizona Medical Association, Inc. held at 810 West Bethany Home Road, Phoenix, Arizona, on Saturday, October 6, 1979, convened at 10:12 a.m., William E. Crisp, M.D., President and Chairman presiding.

Minutes

In reviewing the minutes of the meeting held August 4, 1979, the Board's attention was called to the classification change requested and granted for Sherwood Burr, M.D. — Active to Associate - Account Retirement -Dues Exempt -Effective 1/1/79 — and the fact that there existed some doubt as to whether or not Dr. Burr had, in fact, retired from practice. Although the minutes of the meeting held on August 4, 1979 were correct, in that the Board had granted such change at the request of the Pima County Medical Society and the minutes, therefore, were approved as distributed, the Board took the following action:

It was moved and carried that the Association inquire of the Pima County Medical Society as to whether or not Sherwood Burr, M.D., had retired from active practice significantly enough to justify the change in status, i.e., active to associate, granted by the Association in meeting of the Board of Directors on August 4, 1979.

Auxiliary Activites Report

Mrs. George Hoffman, as a representative from the ArMA Auxiliary, reported on Auxiliary activities which included the newly-established monitoring of Health Services Agencies; the recently held fall conference, at which representatives of the California Medical Association Auxiliary were present; a "Who Are We?" questionnaire which will shortly be sent to the wives of all physicians in the state, with the information gathered to be used in the development and planning of future activities; and the current activites being carried on by the Public Relations Committee.

Board of Directors

Medical Student Representative

Dr. Crisp introduced and welcomed Maureen Mangotich, Co-Chairman, Arizona Chapter, American Medical Students Association, who will be a regular guest at future Board of Directors' meetings representing the medical students.

Resolution Re: Cardio-Pulmonary Resuscitation Training

The Board reviewed and discussed the request dated August 24, 1979 from Dermont W. Melick, M.D. relating to the introduction of a resolution regarding Cardio-pulmonary Resuscitation training at the meeting of the House of Delegates in April, 1980.

It was moved and carried to receive for informational purposes the letter of Dermont W. Melick, M.D. dated August 24, 1979 together with his proposed resolution Re: Cardio-pulmonary resuscitation training and advise Dr. Melick that he could, should he so desire, arrange for the introduction of the resolution at the 1980 meeting of the House of Delegates.

IRS/Malpractice Insurance Surplus Contribution Issue

The Board was informed that legal counsel's opinion relating to the IRS malpractice insurance surplus contribuiton issue was not yet complete and that the matter would be deferred until a future meeting.

Executive Committee

Membership Classification Changes Approved

Maricopa County Medical Society

1. Merlin W. Kampfer, M.D. - Active to Associate -Account Military - Dues Exempt - Effective 1/1/80.
2. Yukio Miyauchi, M.D. - Active to Active Over 70 -Account Age - Dues Exempt - Effective 1/1/80.

Pima County Medical Society

1. Arnold P. Friedman, M.D. - Active to Active Over 70 - Dues Exempt - Effective 1/1/80. -
2. Robert Gearhart, M.D. - Active to Active Over 70 -Account Age - Dues Exempt - Effective 1/1/80.
3. Frederick J. Lesemann, Jr., M.D. - Active to Associate - Account Retirement - Dues Exempt - Effective 6/1/79.
4. George W. Nash, M.D. - Active to Associate - Account Out of Country - Dues Exempt - Effective 1/1/80.
6. Sidney Werner, M.D. - Active to Active Over 70 -Account age - Dues exempt - Effective 1/1/80.
7. Walter Work, M.D. - Active to Active Over 70 -Account Age - Dues Exempt - Effective 1/1/80.

Student Memberships Approved

John E. Bovlet
Donald Buckly
Andrew Epstein
Scott Hitt
Dick Leff
Maureen Mangotich
Sheldon Marks
Richard Moos
Bruce Parks
Ken Patick
Sharon Sandell
Elizabeth Smoots
Diana Stockton
Denny Thrasher
Nettie White
Del Worthington

Ratification of Executive Committee Action

The Board discussed what was felt to be a necessity for the Board of Directors to ratify or approve Executive Committee action on certain matters and specifically action taken by the Executive Committee in meeting on September 7, 1979, relating to the Association's borrowing resolution with the Valley National Bank of Arizona.

It was moved and carried that the Board of Directors of ArMA ratifies and approves the action of the Executive Committee taken in meeting on September 7, 1979, relating to the execution by Association officers of the borrowing resolution for a determined line of credit with the Valley National Bank of Arizona.

It was further moved and carried that all future Board of Directors' agendas shall include, under the Executive Committee category, an item designated "Ratification of Executive Committee minutes."

AMA Delegation

Reports - AMA Meeting

Reports prepared by W. Scott Chisholm, Jr., M.D. and Arthur V. Dudley, Jr., M.D. of the 7/22-7/26/79 AMA Meeting. *Received.*

Nominations for AMA Councils

AMA letter dated August 22, 1979 relating to recommendations for positions on Councils of the AMA received for future action by the Board.

Nominations for Awards

AMA letter dated August 16, 1979 relating to nominations for the Sheen, Beaumont, and Goldberger Awards received for future action by the Board.

Principles of Medical Ethics

The Board reviewed and discussed the Review and Comment on the Report of the Ad Hoc Committee on the Principles of Medical Ethics of the AMA, together with a proposed amended version of the Principles of Medical Ethics. Following a lengthy discussion and comment by the Associations' AMA Delegation.

It was moved and carried that the review and comment on the report of the Ad Hoc Committee on the Principles of Medical Ethics of the AMA, together with a proposed amended version of the Principles of Medical Ethics, would be brought back to the Board of Directors of ArMA at their meeting scheduled for January 5, 1980, with the intention of developing a resolution on the subject matter for presentation to the House of

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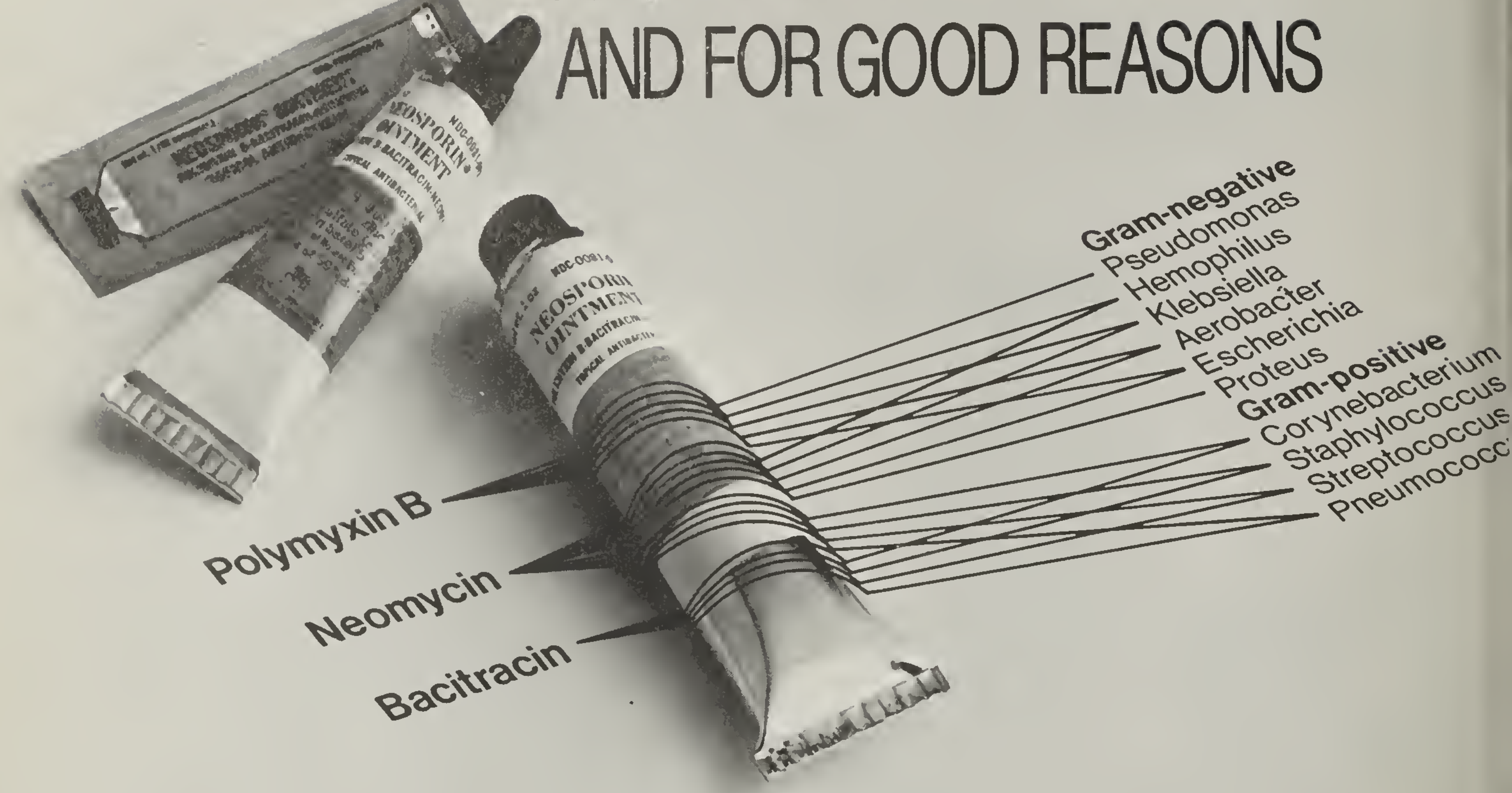

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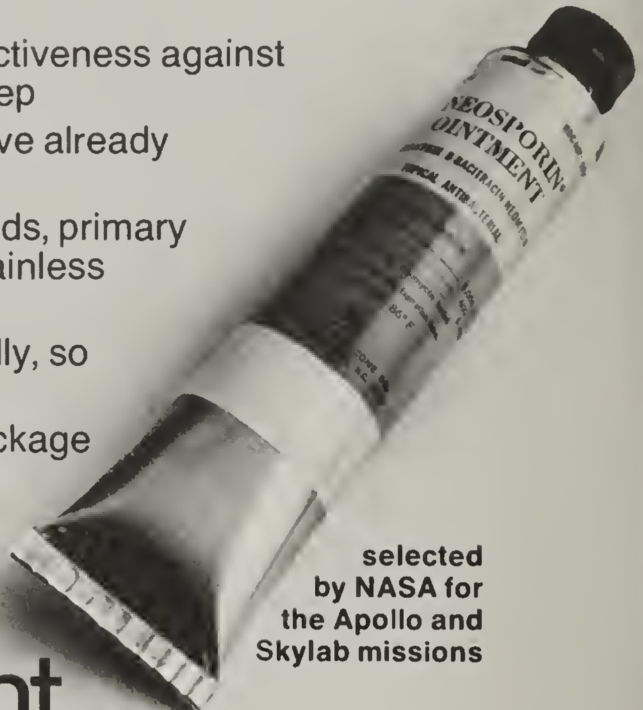

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WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as a failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

Wellcome
Burroughs Wellcome Co.
Research Triangle Park
North Carolina 27709

Delegates in April, 1980.

Benevolent and Loan Fund Committee

Letter of appreciation from medical student, Theresa Cullen. *Received.*

Finance Committee

1980 Committee Budget

The Board considered the recommendation of the Finance Committee relating to the establishment of the 1980 Committee Budgets within the limits set by the House of Delegates in April, 1979, and following discussion:

It was moved and carried to approve the 1980 committee budgets as follows:

PICK UP FROM NOVEMBER

Annual Meeting Registration Fees

The Board considered the actions of the Finance Committee and of the Scientific Assembly Committee, both taken in meeting on September 29, 1979, relating to member registration fees for the annual meeting of ArMA, and after a thorough discussion, took the following action:

It was moved and carried that there be no change in the present system used for registration of ArMA members for attending the Arizona Medical Association Annual Meeting.

Advisory Council on Nursing Manpower & Education Needs

The request for funding from the Advisory Council on Nursing Manpower & Education Needs was again considered by the Board, in conjunction with the recommendation of the Finance Committee relating thereto dated October 2, 1979.

It was moved and carried that the Arizona Medical Association approve, in principle, the study intended to be undertaken by the Advisory Council on Nursing Manpower & Education Needs relative to the shortage of nurses within the State, but that no funds are available within the 1979 budget to offer monetary assistance.

Financial Statement

The Financial Statement for the period ending 8/31/79. *Received.*

MICA/ArMA Contract Negotiations

Dr. Crisp advised the Board that the committees for MICA and ArMA have gotten together and a new contract between the two organizations is in the process of being developed.

Governmental Services Committee

Confirmation of Appointment

It was moved and carried confirming the appointment of Del Worthington, student, to the Governmental Services Committee for the term 1979-82.

Workshop on Health Planning

Gary Barnett reported to the Board that the workshop on Health Planning is scheduled to be held on January 19, 1980; that solicitations for participation had been extended to, among others, the Chariman of the Ad Hoc Health Planning Committee of the AMA and Executive Directors of HSA's; and that, upon confirmation of a complete program, invitations would be prepared and mailed to potential attendees.

Health Manpower Committee

Confirmation of Appointment

It was moved and carried confirming the appointment of Nettie White, student, to the Health Manpower Committee for the term 1979-82.

Podiatrists on Medical Staffs

The Board received and reviewed the recommendation contained in the September 7, 1979 letter from the Health Manpower Committee, together with the pending legislation which would mandate the admission of podiatrists into hospital medical staff. Simultaneously, the Board reviewed the letter directed to Chiefs of Staff and administrators of Arizona Hospitals dated 8/13/79 and the Medical Memos of that same date forwarded to ArMA members reaffirming the joint position paper adopted in 1977 and reaffirmed in 1979 realting to podiatrists and medical staff privileges. After lengthy discussion.

It was moved and carried that a letter be prepared by the Association directed to the Chiefs of Staff, Chairmen of Board of Trustess and/or chief operating officers of hosptials indicating the urgency of consideration of opening medical staffs to qualified Podiatrists, and calling attention to the pending legislation that would mandate the admission of podiatrists into hospital medical staff, thereby allowing no further options in the matter.

Proposed Position Papers

Dr. Linkner reviewed for the Board the position papers which the committee was recommending for adoption as the position papers of ArMA relating to Chiropractic, Naturopathy, Nurse Practitioner and Podiatry; additionally, Dr. Linkner reviewed with the Board legal counsel's letter dated 9/24/79 relative to such position papers; and, following a lengthy discussion,

It was moved and carried that the Arizona Medical Assocation adopt those position papers relating to Chiropractic, Nauropathy, Nurse Practitioner and Podiatry attached hereto and by this reference incorporated as a portion of these minutes. (to conserve space these position papers will not be reprinted, a copy can be obtained by calling the ArMA offices 246-8901).

Legislative Committee

Confirmation of Appointments

It was moved and carried confirming the appointments of Maureen Mangotich and Scott Hitt, students, to the Legislative Committee for the term 1979-82.

Malpractice Insurance Crisis Committee, Ad Hoc

Consumer and Physician Attitudes Toward Physician Services, Medical Malpractice and Patients Study

Following a review of the study prepared by Stephen W. Brown, D.B.A., entitled "Consumer and Physician Attitudes toward Physician Services, Medical Malpractice and Patients" and the recommendations of the Ad Hoc Malpractice Insurance Crisis Committee forumulated in meeting on September 11, 1979.

It was moved and carried that the study, "Consumer and Physician Attitudes Toward Physicians Services, Medical Malpractice and Patients," be published in Arizona Medicine in serial form for a five or six month period.

It was further moved and carried that the Association procure, on a contract basis, the services of Stephen W. Brown D.B.A., and with the assistance of the Association's Public Relations Committee, attempt to develop programs utilizing the study on Consumer and Physician Attitudes toward Physician Services, Medical Malpractice and Patients for appropriate presentations to physician staffs throughout Arizona.

The Board was informed by the Chairman of the Ad Hoc Malpractice Crisis Committee, Dr. Sattenspiel, that the study had been accepted by the Scientific Assembly Committee in meeting on September 29, 1979, for a panel presentation during the scientific portion of the Association's 1980 Annual Convention.

Maternal & Child Health Care Committee

Confirmation of Appointment

It was moved and carried confirming the appointment of Del Worthington, student to the Maternal & Child Health Care Committee for the term 1979-82.

Annual Report - Arizona Perinatal Program

The annual report for the fourth year of the Arizona Perinatal Program. *Received.*

Medical Economics Committee

Confirmation of Appointments

It was moved and carried approving the appointments of Andy Epstein, Sheldon Marks and Don Buckley, students, to the Medical Economics Committee for the term 1979-82.

Life Insurance Program

The Board reviwed the letter from the Medical Economics Committee setting forth a proposal for a new life insurance program for members through the Fireman's Fund, American Life Insurance Company and, following a discussion of the matter,

It was moved and carried that the Association sponsor to its members the life insurance program offered by Fireman's Fund, American Life Insurance Company.

Chairman of Committee

The Board was advised of the resignation of Richard W. Switzer, M.D., as Chairman of the Medical Economics Committee and that Clyde W. Kurtz, M.D. had expressed a willingness to accept such chairmanship for the balance of the unexpired term.

It was moved and carried confirming the appointment of Clyde W. Kurtz, M.D. as chairman of the Medical Economics committee for the term 1979-1980 and as a member of the committee for the term 1979-82.

Medical Education Committee

Confirmation of Appointment

It was moved and carried confirming the appointment of Sharon Sandell, student, to the Medical Education Committee for the term 1979-1982.

Membership Committee, Ad Hoc

Letter dated 8/17/79 from Hoyt Gardner, M.D., President of the AMA. *Received.*

Professional Committee

Confirmation of Appointments

It was moved and carried confirming the appointments of Sheldon Marks and Del Worthington, students, to the Professional Committee for the term of 1979-82.

Public Relations Committee

Confirmation of Appointments

It was moved and carried confirming the appointment of Mrs. Donald Davis, as Chairman of the ArMA Auxiliary Communication/Public Relations Committee, and the appointments of Scott Hitt and Diana Stockton, students, to the Public Relations Committee for the term 1979-82.

Scientific Assembly Committee

Confirmation of Appointment

It was moved and carried confirming the appointment of Dan Morris, M.D., as a Housestaff representative, to the Scientific Assembly Committee for the term 1979-82.

1980 Annual Meeting

General program schedule for the 1980 Annual Meeting. *Received.*

Other Business

Surgicenter

Recognition of Surgicenter by the Health Insurance Institute. *Received.*

Western Physician Purchasing Association

The Board considered the September 6, 1979 request of the Arizona Podiatary Association that its members be allowed to participate in the Western Purchasing Association, and after a lengthy discussion,

It was moved and carried to table the request of the Arizona Podiatry Association regarding participation by it members in the Western Physicians Purchasing Association until the President of WPPA, John F. Kahle, M.D., could be present at a meeting of the Board of Directors and address the matter in depth.

The IPA - ArMA's Role in Development

Dr. Crisp addressed the Board regarding the HMO's presently existing throughout the state and any advantages which might be gained by the attempt to establish an Independent Practice Association in order to compete. Additionally, Dr. Laurance B. Nilsen spoke regarding the recent discussions by the Foundation relating to similar considerations. Following a lengthy discussion, it was the consensus of the Board that the Association should make no efforts in the establishment of an IPA.

MATERNAL AND CHILD HEALTH CARE COMMITTEE

The meeting of the Maternal and Child Health Care Committee of the Arizona Medical Association, Inc. held at 810 West Bethany Home Road, Phoenix, Arizona, on Saturday, October 13, 1979, convened at 10:10 a.m., William C. Scott, M.D., Chairman, presiding.

Perinatal Mortality Review

The committee discussed at length and in great detail the need for an feasibility of a Statewide Perinatal Mortality Review based upon a proposal presented by Dr. William Carlile for review, after which

It was moved and carried to establish and Ad Hoc Committee to Review the Proposal for a Statewide Perinatal Mortality Review Committee, directing such committee to return to the next meeting of the Maternal and Child Health Care Committee with specific objectives for such review and guidelines by which it could be implemented.

Midwifery

Status Report

Dr. Carlile briefly addressed the committee regarding the activities of the Midwife Licensing Advisory Committee, who have been very active and are in the process of establishing guidelines for the practice of midwifery.

Reporting Forms

It was brought to the committee's attention that, although forms for reporting out-of-hospital complications had been forwarded several months ago to various hospitals within the state, few forms had actually been completed and returned. Following a discussion, it was the consensus that the Chairman send a follow-up letter to the Chiefs of Service of those hospitals initially contacted, requesting them to again discuss the reporting form with their staff and encouraging their cooperation in completing and returning the forms.

Licensed Midwife Data

Licensed Midwife Data Sheet for calendar year 1978. *Received.*

H.B. 2336 - Maternity Benefits

Letter of 5/10/79 to Walter B. Cherny, M.D., as Chairman of Subcommittee on Maternal Health. *Received.*

Dr. Richard O. Flynn, as chairman of the Legislative Committee, addressed the committee regarding H.B. 2336 which provides, among other things, the inclusion of maternity benefits in certain insurance policies, stating that, although this had been presented to the legislature for many years, it would probably continue to fail until some prediction as to cost could be made.

Registered Nurse Practitioners

The committee received and reviewed a proposal from the Phoenix OB/GYN Society and the Arizona Gynecological Nurse Practitioner Association for legislation relating to the "Licensing and Practice of Registered Nurse Practitioners." After a lengthy discussion, the committee took the following action:

It was moved and carried that the Maternal and Child Health Care Committee supports, in concept, the proposed legislation relating to "Licensing and Practice of Registered Nurse Practitioners" submitted by the Phoenix Obstetrical-Gynecologiccal Society and the Arizona Gynecological Nurse Practitioner Association, a copy of which is attached to these minutes and by reference in coporated herein. (to conserve space the legislation will not be reprinted, a copy can be obtained by calling the ArMA offices 246-8901.)

It was further moved and carried to refer the proposed legislation to the Legislative Committee for its review and coonsideration, with the suggestion that the joint committee therein referred to be changed to a composition of three medical doctors, three registered nurses, one pharmacist and one consumer.

Postion Paper Re: Nurse Practitioners adopted by ArMA Board of Directors on 10/6/79. *Received.*

Arizona Perinatal Program

Annual Report - Fourth Year

The Fourth Annual Report of the Arizona Perinatal Program was received and reviewed by the committee. Additionally, Dr. Harold Gabel, Executive Director of APP, discussed briefly the achievements of the past year, as well as the goals for the year to come.

Other Business

Genetics Section

Memorandum dated 7/18/79 to Harold D. Gabel, M.D., as Chairman of the Section on Genetics. *Received.*

Appointment

Board of Directors' confirmation of appointment of Del Worthington, student, to committee. *Received.*

PHYSICIAN REHABILITATION COMMITTEE

The meeting of the Physician Rehabilitation Committee of the Arizona Medical Association, Inc. held at 810 West Bethany Home Road, Phoenix, Arizona, on Wednesday, October 17, 1979, a quorum being present, convened at 6:13 p.m. Thomas E. Bittker, M.D., Chairman, presiding.

Alcoholism & Mental Health Insurance Coverage

The Chairman presented for the committee's review a proposal for consideration by the Association as its position relative to mental health insurance coverage. Following a lengthy discussion.

It was moved and carried to recommend to the Board of Directors of ArMA that the Association adopt, as its position relative to mental health insurance coverage, the following:

"The Arizona Medical Association recognizes that alcoholism, drug abuse and emotional disorders represent significant threats to the health of physicians and their families. These are all treatable problems given the availability of early intervention and comprehensive medical treatment. The Association urges that all physicians have available to them health insurance policies that facilitate the treatment of these disorders. Although health insurance may include reasonable deductibles to control cost of care, the Association believes that deductibles charged for treatment of these disorders should be non-discriminatory—no greater than those charged for other medical conditions.

The Association recommends further that any health insurance policies offered by the Association or its constituent county societies provide comprehensive coverage for the treatment of alcoholism, drug abuse and emotional disorders for physicians and their families. Although specific insurance policy guidelines should be determined by the purchasing group, a model policy would include coverage up to 45 outpatient visits per year and 30 inpatient days per year, at a co-insurance of no greater than 20% for the treatment of these disorders.

New Reporting Form

A new reporting form, incoporating data from case assignment through final follow-up status, was reviewed and discussed by the committee, and

It was moved and carried to adopt the new reporting form (Copy of which is attached hereto and by this reference incoporated herein) for use by the members to the committee. (to conserve space the form will not be reprinted, a copy can be obtained from the ArMA offices 246-8901.)

It was determined that a set of sample forms would be immediately forwarded to committee members and that additional forms would be available by request from the Association offices.

Regional Meeting - Impaired Physician Program

Dr. Bittker reported on his recent attendance of a regional meeting held in San Francisco relating to the impaired physician program, which consisted of representatives of all Western states, including Hawaii. It appeared that all represented states had concerns similar to ours and some of the issues of general discussion were concern that regionalized meetings should not supplant national conferences; the desire to make a more concerted effort to involve recovered physicians in state/regional programs; and a "physician only" inpatient service.

A lengthy discussion ensued relative to the recovered physician and the necessity and desire to attempt to organize a recovered physicians force to assist in the aiding of impaired physicians; and it was determined that a subcommittee, consisting of Harrison G. Butler, M.D., as chairman, Thomas E. Bittker, M.D., Paul B. Borgeson, M.D., B. Rossd Landess, M.D. and Louis E. Young, M.D., be formed to make initial contact with recovered physicians and report back to the next committee meeting with recommendations as to the feasibility of such a group being formed.

Other Business

Issue of Confidentiality

The committee discussed at great length the concern of the psychiatric community regarding the issue of confidentiality and the present statutes requiring reporting of allegedly impaired physicians, particularly as it relates to cases wherein a psychiatrist may be a provider of services to such individual. It was the consensus of opinion that the obligatory reporting law should be amended and that the committee should initiate such action through the Association and the Arizona Psychiatric Society and, thereafter,

It was moved and carried to recommend to the Board of Directors of the Arizona Medical Association and to the Arizona Psychiatric Society that a joint committee be formed for the purpose of reviewing the existing state obligatory reporting laws regarding an impaired physician with a view to drafting legislation which would amend such statutes to apply to all physicians **except** *those physicians who are involved in a provider relationship with the alleged impaired physician; and that, in those instances, except when there exists imminent threat of harm to self or others, physician/patient privilege takes priority and the care-taking physician is thus exempt.*

MEDICAL EDUCATION COMMITTEE

Meeting of the Medical Education Committee of the Arizona Medical Association, Inc., held on Thursday, November 8, 1979, at 810 West Bethany Home Road, Phoenix, convened at 6:55 p.m., Robert E.T. Stark, M.D., Chairman, presiding.

Introduction

Members were introduced and Ms. Sharon Sandell, medical student, was welcomed as a new member.

Section Reports

Section on Accreditation: - William F. Sheeley, M.D., Chairman

Doctor Sheeley reported that the Section on Accreditation had met and recommends approval of the reports of the survey teams who had visited the Arizona Division, American Cancer Society; Walter O. Boswell Memorial Hosptial and Health Maintenance Associates.

It was moved and carried to grant provisional accreditation of the Arizona Division, American Cancer Society for one year with written progress report in six months and re-survey in one year.

It was moved and carried that Walter O. Boswell Memorial Hospital have provisional accreditation for one year with a written progress report in six months and re-survey in one year.

It was moved and carried that Health Maintenance Associates be re-accredited for a period of four years with the stipulation that they present a *written progress report to this committee in two years.*

It was suggested that in future surveys an attempt be made to coincide the site visit with a program that is being carried out at the institution or organization applying for accreditation.

The Section considered an accreditation application from Northern Arizona Medical Evaluation System (NAMES) and decided to request further information from NAMES as to the type of CME program they are presenting.

It was recommended that survey teams be dispatched for survey of the Arizona Thoracic Society/Arizona Lung Association, and re-survey of the Arizona State Hospital.

Section on Certification: - Ashton B. Taylor, M.D., Chairman

1977

Doctor Taylor reported that the Section on Certification had met and considered the request of a physician who is delinquent in reporting his CME activities due in 1977. Due to extenuating circumstances described in correspondence, an extension was granted until January 1, 1980.

1978

Regarding those who have not as yet reported their activities for the 1978 period, it was decided that members of this Section, as well as those of the Medical Education Committee, personally contact those known to them. Also, a certified letter is to be sent to these physicians advising that unless their 1978 CME Application is received by December 31, 1979, it will be recommended to the Board of Directors of the Arizona Medical Association that they be dropped from membership.

1979

It was reported that there still remain 337 members due to report this year who have not done so.

It was moved and carried to approve the recommendations of the Section on Certification.

Section on Requirements: - Albert G. Wagner, M.D., Chairman

Doctor Wagner reported on the co-sponsorship program and again emphasized the need for close liaison between sponsoring organizations and the non-accredited programs that are being co-sponsored.

Reports were received from several organizations for whom ArMA has co-sponsored programs and are as follows:

1. A written report from the Arizona Perinatal Program with copies of brochures on their recent CME Programs.
2. Written summary from Alan C. Winfield, M.D. of Category 1 CME Programs carried out at the Yuma Regional Medical Center.
3. Alex Newman, M.D. reported that the Phoenix Radiological Society had planned ten meetings for the period of September, 1979 through May, 1980, including a joint meeting with the Phoenix Society of Gastroenterology.

Written request of 10/25/79 from Thomas E. Vorpahl, M.D. was reviewed. He is requesting co-sponsorship of CME programs of Flagstaff Community Hospital so that they may receive Category 1 credit. He has volunteered to serve on the Medical Education Committee so that he may act as liaison. Attached to his letter were listings of current and planned program. The President of ArMA will be asked to appoint Doctor Vorpahl to the committee.

AMA/LCCME Split

Excerpt from *CME Marketing Memo* of September, 1979, announcing that AMA withdrew from LCCME on July 25, 1979, was reviewed. LCCME, composed of representatives from numerous medical specialties, announced that it would continue to function as a CME accrediting body. Letter from the LCCME dated October 23, 1979, asking that they be informed of future accreditation actions of ArMA was reviewed. Also received and discussed was a letter from AMA dated October 30, 1979, stating that LCCME had asked for all records of accreditation actions for the period of July, 1977 - July, 1979, now in AMA's possession, and requesting that ArMA advise AMA as to our wishes in the matter. After considerable discussion it was agreed to inform LCCME of our accreditation activities at the time a report is sent to AMA, and also to advise AMA we have no objections to their releasing accreditation information to LCCME.

It was moved and carried that we send to the LCCME a copy of information provided AMA regarding accreditation actions of ArMA.

NCSCCME

Letter from Melvin Small, M.D., Chairman of the National Council of State Committees of Continuing Medical Education (NCSCCME) dated 9/14/79 regarding membership in this institution was distributed. After discussion concerning continuation of membership in the NCSCCME, the following action was taken:

It was moved and carried not to maintain membership in the NCSCCME.

AMA CME Conference

In the absence of Robert E. Hastings, M.D. who was to have presented a report, Misty Coumbe gave a verbal report on the CME Conference in Chicago which took place on October 18 and 19, 1979.

AAFP Actions

Article from *Medical World News* reporting on AAFP actions concerning categories of credit was received for information.

ArMA CME Calendar

A letter from Thomas J. Conahan, III M.D. expressing appreciation for the calendar was received for information.

Academy of Medicine

Several members who are also involved with activities of the CME program of the Maricopa County Medical Society Academy of Medicine reported on the progress of that group's planning. It was stated that current planning seems centered around development of a new facility for the Robert S. Flinn Medical Library.

Search for Director of CME & Outreach at U of A

In response to a request from the University, ArMA has appointed a committee to interview candidates for the position. Those appointed are: Richard S. Armstrong, M.D., John T. Clymer, M.D. and W. David Jacoby, M.D.

Other Business

Arnold Serbin, M.D. recommended consideration of a physician in private practice sitting on the Admissions Committee at the University of Arizona College of Medicine. After considerable discussion it was suggested that a letter be written to the medical school indicating an interest in having a physician in private practice on the Admissions Committee of the medical school.

It was moved and carried that a letter be sent to the U. of A. College of Medicine suggesting that physicians in private practice be represented on the admissions committee.

Future Medical Meetings

The following institutions and organizations have been accredited for their continuing medical education programs by the Liaison Committee on Continuing Medical Education

- Arizona Chapter, American Cancer Society
- Arizona Heart Institute
- Arizona Medical Association
- Arizona State Hospital, Phoenix
- Camelback Hospital, Phoenix
- Desert Samaritan Hospital, Mesa
- Good Samaritan Hospital, Phoenix
- Health Maintenance Associates, Phoenix
- Maricopa County General Hospital, Phoenix
- Memorial Hospital of Phoenix
- Mesa Lutheran Hospital, Mesa
- Phoenix Baptist Hospital & Health Center
- Phoenix Indian Medical Center
- Phoenix Psychiatric Council
- St. Joseph's Hospital & Medical Center, Phoenix
- St. Lukes's Hospital & Medical Center, Phoenix
- St.Mary's Hospital, Tucson
- Scottsdale Memorial Hospital
- Tucson Hospitals Medical Education Program, (THMEP) Tucson
- University of Arizona College of Medicine, Tucson
- Veterans Administration Medical Center, Phoenix
- Veterans Administration Hospital, Prescott

The accredited institutions and organizations above produce a variety of continuing medical education programs. Each accredited institution and organization is responsible for designating which of these programs meet ArMA's requirements for Category 1 credit. Physicians who participate in programs which are designated Category 1 by accredited institutions will receive Category 1 credit toward the ArMA Certificate in CME and the AMA's Physician's Recognition Award.

1980

JANUARY

Impotence
January 9, 1980. Maryvale Samaritan Hospital. Sponsor: Good Samaritan Hospital. Contact: CME office, Good Samaritan Hospital, 1033 E. McDowell Road, Phoenix, AZ 85006. Approved for 1 hour of category 1 credit.

Evaluation & Treatment of Coagulation Disorders
January 10, 1980. Williams Air Force Base, Hospital Conf. Rm. Sponsor: USAF Manpower & Personnel, Surgeon's Office. Contact: Williams Air Force Base, Chandler, AZ. Approved for 1 hour of category 1 credit.

OBGYN Seminar
January 10-11, 1980. Arizona Health Sciences Center, Tucson, AZ. Sponsor: U of A College of Medicine. Contact: Office of CME and Outreach, Arizona Health Sciences Center, Tucson, AZ 85724

General Pediatrics #1
January 10-12, 1980. Doubletree Inn, Scottsdale, AZ. Sponsor: American Academy of Pediatrics. Contact: Ms. Judy C. Hambourger, Dept. of Education, American Academy of Pediatrics. P.O. Box 1034, Evanston, Ill 60204. Approved for 18 hours of category 1 credit.

Diagnosis & Treatment of Headache
January 11-13, 1980. La Posada, Scottsdale, AZ. Sponsor: American Association for Study of Headache. Call: Robert Kunkel, M.D. (216) 444-5654. Approved for 15½ hours of category 1 credit.

Case Studies In Emergency Care
January 12, 1980. The Adams Hotel, Pheonix, AZ. Sponsor: U of A College of Medicine. Contact: Office of CME and Outreach, Arizona Health Sciences Center, Tucson, AZ 85724. Approved for 7 hours of category 1 credit.

Advanced Cardiac Life Support Program (Provider Course)
January 11-12, 1980. Arizona Medical Association, Phoenix, AZ. Sponsor: ArMA & Arizona Affiliate - American Heart Assoc. Contact: Carol Tuner, ArMA Offices 810 W. Bethany Home Road, Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

23rd. Annual Cardiac Symposium
January 18-19, 1980. Mountain Shadows Resort, Scottsdale, AZ. Sponsor American Heart Association & Maricopa County General Hospital. Call: Charlotte Beeler 277-4846.

Imaging Modalities for the Practicing Physician
January 19, 1979. Education Building, Good Samaritan Hospital, 1033 E. McDowell Rd., Phoenix, AZ 86006. Sponsor. Good Samaritan Hospital. Contact: CME office at Good Samaritan Hospital. Approved for 6 hour of category 1 credit.

Introduction to Neuropsychological Evaluation & the Halstead-Reitan Test Battery.
January 19, 1979. Hilton Hotel, Scottsdale, Az. Sponsor: St. Luke's Hospital & Medical Center, 525 N. 18th St., Phoenix, AZ 85006. Contact: CME Office St. Luke's Hospital. Approved for 6 hours of category 1 credit.

8th Annual Surgical Symposium
January 21-25, 1980. Doubltree Inn, Scottsdale, AZ. Sponsor: Maricopa County General Hospital, & Phoenix Surgical Society. Call: Susie Davis 264-9021. Approved for 31 hours of category 1 CME credit.

Recognition of Problems of the Hand
January 23, 1980. Glendale Samaritan Hospital, Glendale, AZ. Sponsor: Good Samaritan Hospital. Contact: Good Samaritan Hospital Office of CME, 1033 E. McDowell Rd., Phoenix, AZ 85006.

Medical Aspects of Family Planning
January 24, 1980. Williams Air Force Base, Hospital Conf. Rm. Sponsor: USAF Manpower & Personnel Center & Surgeon's Office. Contact: Williams Air Force Base, Chandler, AZ. Approved for 1 hour of category 1 credit.

Continuing Education In Perinatal Medicine
January 25-26, 1980. Yuma, Arizona. Sponsor: ArMA & Arizona Perinatal Program. Contact: Arizona Perinatal Program, 810 W. Bethany Home Road, Phoenix, AZ 85013. Approved for 15 hours of category 1 credit.

Sleep Disorders
January 26, 1980. Good Samaritan Hospital, Phoenix, AZ. Sponsor: Good Samaritan Hospital. Contact: GSH, CME office, 1033 E. McDowell Road, Phoenix.AZ 85006. Approved for 6 hours of category 1 credit.

27th Annual Scientific Assembly of AAFP
January 20-Feb.2, 1980. Doubletree Inn, Scottsdale, AZ. Sponsor: U of A College of Medicine, & AAFP. Call: Lee Mahoney 949-7797. Approved for 19 hours of category 1 credit.

Medical History Society of Arizona
January 31, 1980. Maricopa County Medical Society, Preston T. Brown Room, 2025 N. Central Ave., Phoenix, AZ 85004. Contact: William C. Weese, M.D., 265-0000.

FEBRUARY

2nd Annual Southwest Desert Symposium In Anesthesiology
February 1-3, 1980. Doubletree Inn, Tucson. Sponsor: U of A College of Medicine. Contact: Office of CME & Outreach, AHSC, Tucson, AZ 85724.

Meeting of the Society of Cardiovascular Radiology
February 3-7, 1979, Hyatt Regency Hotel, Phoenix, AZ. Sponsor: Society of Cardiovascular Radiology. Contact: Wm. Casarella, M.D. (212) 694-2286. Approved for 23 hours of category 1 credit.

Osteomyelitis-Causes, Diagnosis and Treatment February 7, 1980. Williams Air Force Base, Hospital Conf. Romm. Contact: USAF Manpower & Personnel Center, Surgeon's Office. Approved for 1 hour of category 1 credit.

Annual BNI Symposium - "New Perspectivies In Cerebral Localization"
February 7-9, 1980. Hyatt-Regency Hotel, Phoenix, AZ. Sponsor: St. Joseph's Hospital and Medical Center, Barrows Neurological Institute. Contact: Richard A. Thompson, M.D., Chairman, 350 West Thomas Rd., Phoenix, AZ 85013.

Emergency Care Update
February 7-9, 1980. Arizona Health Sciences Center, Tucson. Sponsor: U of A College of Medicine. Contact: Office of CME & Outreach, AHSC, Tucson, AZ 85724

Coronary Disease, Exercise Testing & Cardiac Rehabilitation
February 8-10, 1980. Mountain Shadows Resort, Scottsdale, AZ. Contact International Medical Education Corp., 64 Inverness Drive East, Englewood, Co 80112. Approved for 13 hours category 1 credit by AMA, and 13 prescribed hours by AAFP

Advanced Cardiac Life Support Program (Provider Course)
February 8-9, 1980. ArMA Offices, 810 W. Bethany Home Rd., Phoenix, AZ 85013. Sponsor: A.C.E.P., AZ Affiliate American Heart Assoc. & ArMA. Contact: Carol Turner, ArMA offices. Approved for 16 hours of category 1 credit.

Medical Controversies
February 8-10, 1979. Registry Resort, Scottsdale, AZ. Sponsor: Health Maintenance Associates. Contact: Clifford Harris, M.D., 957-9200.

5th Annual Seminar - Radiology & Geriatric Medicine
February 8-10, 1980. Doubletree Inn, Scottsdale, AZ. Sponsor: Maricopa County General Hospital. Contact: Austin R. Sandrock, M.D., Program Chairman. M.C.G.H., Dept. of Radiology, 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 15 hours of category 1 credit.

Myeloproliferative Syndromes
February 11-15, 1980. Del Webb's Townhouse, Phoenix, z. Sponsor: American Society of Clinical Pathologists. Contact: American Soc. of Clinical Path., 2100 W. Harrison St., Chicago, Ill. 60612. Approved for 30 hours of category 1 credit.

Taos Medical Symposium
February 12-15, 1980. Tennis Ranch of Taos, Taos, New Mexico. Sponsor: New Mexico Chapter, AAFP, CME Committee, New Mexico Medical Society, Albuquerque Academy of Family Physicians, Albuquerque & Bernalillo County Medical Association. Contact: Thomas A. Bodnar, Program Director, 2650 Yale Blvd., S.E., No. 103, Albuquerque, New Mexico 87106

Southwestern Seminar on REhabilitation of Patients with Neuromuscular Disorders
February 14-16, 1980. Arizona Health Sciences Center, Tucson, AZ. Sponsor: U of A College of Medicine. Contact: Office of CME & Outreach, AHSC, Tucson, AZ 85724.

Nineth Annual Day of Medicine: Day of Cardiology
February 15, 1980. Arizona Biltomore, Phoenix, AZ. Sponsor: St. Joseph's Hospital & Medical Center, & Flinn Foundation. Contact: Darlene Graham, Dept. of IM, St. Joesph's Hospital, P.O. Box 2071, Phoenix, AZ 85001. Approved for 6 hours of category 1 credit.

Neurosurgical Problems: Update for the Primary Physician
February 16, 1979. The Adams Hotel, Phoenix, AZ. Sponsor: U of A College of Medicine. Contact: Office of CME & Outreach, AHSC, Tucson, AZ 85724. Approved for 7 hours of category 1 credit.

2nd Annual Ski & Study Program
February 16-21, 1980. Park City, Utah. Sponsor: Desert Samaritan Hospital CME Committee. Contact: Betty Wood, Desert Samaritan Hospital, Mesa, AZ.

Eighth Annual Taos Lung Disease Symposium
February 22-24, 1980. Sponsor: New Mexico Thoracic Society. Contact: New Mexico Thoracic Society, 216 Truman N. E., Albuquerque, N. 87108. Approved for 13½ hours of category 1 credity by AMA.

Second Annual International Cataract Surgery Symposium
February 18-20, 1980. Sponsor: Arizona Foundation for Ophthalmic Research and Education. Contact: Ronald W. Barnet, M.D., Medical Director, 421 N. 18th St., Ste 105, Phoenix, AZ 85006. (602) 257-1077. Approved for 18 to 20 hours of category 1 credit.

3rd International Conf. on Integrated Cancer Management
February 20-22, 1980. The Pointe Resort, Phoenix, AZ. Sponsor: Good Samaritan Hospital. Contact: Office of CME, Good Samaritan Hospital, 1033 E. McDowell Rd., Phoenix, AZ 85006. Approved for 16 hours of category 1 credit.

Evaluation & Management of G-I Bleeding
February 21, 1980. Williams Air Force Base, Hospital Conf. Room. Sponsor: USAF Manpower & Personnel Center & Surgeon's Office. Approved for 1 hour of category 1 credit.

Frontiers in Ophthalmology

February 21-23, 1980. LaPosada Resort, Scottsdale, AZ. Sponsor: Rockefeller & Abbie Prentic Eye Institute of St. Luke's Hospital, 525 N. 18th St., Phoenix, AZ 85006. Contact: Office of CME St. Luke's Hospital. Approved for 15 hours of category 1 credit.

8th Annual Medicine Conference Advances in Internal Medicine II
February 21-23, 1980. Carefree Inn & Resort, Carefree Arizona 85008. Sponsor: Maricopa County General Hospital. Contact: Myra DaBolt, 2601 E. Roosevelt St., Phoenix, AZ 85008. Approved for 21 hours category 1 credit.

Gastroenterology for Clinicians - New Directions
February 21-23, 1980. Del Webb's TowneHouse, Phoenix, AZ. Sponsor: David C.H. Sun Memorial Institute. Contact: Mrs. David C.H. Sun, David C.H. Sun Memorial Institute, 4129 East Sandy Mt. Rd., Scottsdale, AZ 85253.

Advanced Cardiac Life Support (Instructor Course)
February 22-23, 1980. ArMA Offices 810 W. Bethany Home Rd., Phoenix, AZ. 85013. Sponsor: A.C.E.P., AZ Affiliate American Heart Assoc. & ArMA. Contact: Carol Turner, ArMA offices.Approved for 16 hours of category 1 credit.

The Day of Immunology
February 23, 1980. Paradise Valley Country Club, Homestead Rm. Phoenix. Sponsor Scottsdale Memorial Hospital, & St. Luke's Hospital Medical Center. Contact: CME office St. Luke's Hospital Medial Center, 525 N. 18th St. Phoenix, AZ 85006. Aproved for 7 hours of category 1 credit.

Anesthesia for the 80's
February 23-24, 1980. Doubletree Inn Tucson, Az. Sponsor: U of A College of Medicine. Dept. of Anes. Contact: Office of CME and Outreach, AHSC, Tucson, AZ 85724. Approved for 11 hours of category 1 credit.

Pediatric Update 1980
February 25-28, 1980. Sponsor: St. Joesph's Hospital and Medical Center, Dept. of Pediatrics. Contact: Ronald A. Christensen, M.D., Dept. of Pediatric Education. St. Joseph's Hospital and Medical Center, 350 W. Thomas Rd., Phoenix, AZ 85013. Approved for 16 hours of category 1 credit.

Third Annual Mid-Winter Symposium in OB/GYN
February 27- 29, 1980. Scottsdale Hilton Hotel, Scottsdale, AZ. Sponsor: Maricopa County Hospital, Dept. of OB/GYN and Phoenix OB/GYN Society. Contact: The Secretary, Phoenix, OB/GYN Society, 333 East Virginia, Ste 222, Phoenix, AZ 85004.

New Mexico Metabolism Symposium
February 28-March 1, 1980. Taos, New Mexico. Contact: Office of Education. Lovelace Medical Center, Albuquerque, New Mexico. (505) 842-7353.

Contemporary Managment of Stroke and Coronary Disese
February 27 - March 1, 1980. Lodges in Taos, New Mexico. Sponosr: Symposia de Santa Fe. Contact: W.J. Levy, M.D., Symposia de Santa Fe, P.O. Box 5175, Santa Fe, New Mexico 85702

Self-Assessment Program V
February 29, March 1, 1980. Arizona Health Sciences Center, Main Auditorium. Sponsor: U of A College of Medicine & American College of Physicians. Contact: Office of CME and Outreach, AHSC, Tucson, AZ 85724.

MARCH

2nd Annual Symposium on Current Concepts in the Managment of Acute and Chronic Pain
March 1-2, 1980. Hyatt Regency, Phoenix, AZ. Sponsor: Maricopa County General Hospital. Contact: George Wallace, M.D., Program Director, MCGH, 2601 E. Roosevelt, Phoenix, AZ 85008.

Durango, Colorado Ski Symposium
March 5 and March 9, 1980. Sponsor: Albuquerque Academy of Family Physicians. Conact: Albuquerque Academy of Family Physicians, c/o New Mexico Medical Society, 2650 Yale Blvd., S.E., Ste 105, Albuquerque, New Mexico 87106.

Clinical Recognition and Management of Heart Disease
March 27-29, 1980. U of A Health Sciences Center, Main Auditorium. Sponsor: U. of A. College of Medicine & American College of Physicians. Contact: Office of CME and Outreach, AHSC, Tucson, AZ 85724.

Sixteenth Annual Arizona Chest Symposium
March 28-30, 1980. Doubletree Inn, Tucson, Arizona. Sponsor: University of Arizona College of Medicine, and Tucson Medical Center. Contact: Linda Alpert, R.N., Symposium Coordinator, Tucson Medical Center, P.O. Box 6067, Tucson, AZ 85733. Approved for category 1 credit.

APRIL

Emergency Room Radiology-Revised

April 11-13, 1980. Arizona Biltmore, Phoenix, Arizona. Sponsor: Maricopa County General Hospital. Contact: Austin R. Sandrock, M.D., Program Chairman, Maricopa County General Hospital, 2601 E. Roosevelt St., Phoenix, AZ 85008

1980 Third Annual Arizona Emergency Medicine Symposium
April 23-25, 1980, Safari Resort, Scottsdale, Arizona.Sponsors: Arizona Emergency Medical Systems, Inc.; Arizona Chapt Am. College of Emergency Physicians, Arizona Chap. of Emergency Dept. Nurses, Assoc., Arizona Paramedic Cont. Educ. Program. Contact: Jennie Johnston, Symposium Coordinator. 301 W. Osborn Rd., Ste 112, Phoenix, AZ. 85013.

MONTHLY OR WEEKLY

Shrine Medics Meeting
Second Tuesday of each month, Doctors Hospital, 5:45 p.m. J. South Classroom. Sponsor: Shrine Medics. Contact: Robert C. Briggs, M.D., 5121 N. Central Ave., Phoenix, AZ 85012.

Office Psychiatry for the Primary Provider
2nd Monday of month, 4811 N. 7th Street, Phoenix, AZ. Sponsor: Arizona Health Plan. Contact: T.R. Bittker, M.D., Box 5000, Phoenix, AZ 85010. Approved for category 1 credit.

Counter Transference Group
Weekly, Thurs. 8—10 p.m. Sponsor: Phoenix Psychiatric Council. Contact: James E. Campbell, M.D., 5051 N. 34th St., Phoenix, AZ 85018. Approved for 2 hours category 1 credit.

Monthly Meeting of Tucson Radiologists
Last Tues. of month. Plaza International, Tucson, AZ. Sponsor: U of A Medical Center, Dept. of Radiology. Contact: Irwin M. Freundlich, M.D., U of A Health Sciences Center, Dept. of, Radiology, Tucson, AZ 85724. Approved for 2 hours category 1 credit.

Pediatric Grand Rounds
Tuesday 7:30-8:30 a.m in Phoenix: 1st Tues.—Phoenix Indian Hospital, 2nd Tues.—Maricopa County Hospital, 3rd Tues.—Good Samaritan Hospital, 4th Tues.—St. Joseph's Hospital. Sponsor: Maricopa County Hospital (Phoenix Hospital's Affiliated Pediatric Program). Contact: J. Kipp Charlton, M.D., 2601 E. Roosevelt, Phoenix, AZ 85008. Approved for 1 hour per session category 1 credit.

Review of Forensic Pathology Current Case, Special Topics
Thursday, weekly, 11 a.m., 120 S. 6th Ave., Phoenix, AZ. Sponsor: Arizona Society of Pathologists. Contact: H.H. Karnitschnig, M.D., 120 S. 6th Ave., Phoenix, AZ. Approved for 1 hour per session category 1 credit.

ARIZONA HEART INSTITUTE

4800 N. 22nd St., Phoenix, P.O. Box 10,000, Phoenix, AZ 85064. Contact: Edward B. Diethrich, M.D., Medical Dir.

Clinical Conference Cardiovascular Medicine
Third Tues., second floor classroom

ARIZONA STATE HOSPITAL

2500 E. Van Buren, Phoenix, AZ. 85008. Contact: Arnold L. Kendall, M.D.

A.S.H. Psychiatric Grand Rounds
2nd Wed., 1:00-2:00 P.M., J-6 Conf. Rm., Contact: Dr. Conger & Staff
Clinical-Pathological Conference
3rd Wed., 1:30-2:30 P.M. General Services Bldg., Conf. Rm.
Medical Grand Rounds
4th Wed., 1:00-2:00 P.M., Medical Bldg. Conf. Rm.

BARROW NEUROLOGICAL INSTITUTE

Medical Education
Barrow Neurological Institute of St. Joseph's Hospital and Medical Center, 350 W. Thomas Rd., Phoenix, AZ 85013. Sponsor: St Joseph's Hospital & Medical Center. Contact: John R. Green, M.D. Approved for 1 hour category 1 credit.

Neurology Teaching Conference
Tuesdays, 8:30-9:30 a.m., Eighth Floor Conf. Room.
Neurosurgery Morbidity Conference
Wednesdays, 8:15-9:15 a.m., on first and third weeks, Eighth Floor Conference Room.
Neuro-ophthalmology Conference
Tuesdays, 7:30 a.m. in 8th floor neurology conference room.
Spinal Injury Conference
Wednesdays, 8:15-9:15 a.m., on second and fourth weeks, in Neuropathology Conf. Rm.—a multidisciplinary review of admission by neurosurgeons, orthopedists, and rehabilitation specialists
Neuro-opthalmology Conference.
Weekly, 7:30 a.m., 8th floor conference Rm.
Neuropathology of Gross Specimens Conference
Thursday, 7:30-8:30 a.m. in the Morgue.
Neurology-Neurosurgical
Fridays, 8-9 a.m., First Floor Conference Rm.
Neuropathology or Neuroradiology Conferences
Friday, 9 a.m., Neuropathology in Neuropathology Conference Rm., Neuroradiology in First Floor Conference Rm.
Neurorehabilitation Conference
Tuesdays, noon, 8th Floor Conference Rm.
Neurosurgical Journal Club
Saturdays, 9-11 a.m. in Eighth Floor Conference Rm.

CAMELBACK HOSPITAL

5055 N. 34th St., Phoenix, AZ 85018. Sponsor: Camelback Hospital. Contact: Medical Director.

Clinical Conference
3rd Tuesday, 8-9 a.m.

DESERT SAMARITAN HOSPITAL

1400 South Dobson Road, Mesa, Arizona, Contact: L.A. Rosati, M.D. Approved for category 1 credit.

Clinical Conference - Dept. of Medicine
Weekly Thurs., 12:30-1:30 P.M. Buffet Lunch, Grande Rm.
OB/GYN Medical Staff Conference
Weekly, Monday, 12:30-1:30 P.M. Grande Rm.
Pediatric Case Conference
2nd. Friday, 12:30 P.M., Grande 2

EL DORADO HOSPITAL TUCSON, (THMEP)

1400 N. Wilmont Road, Tucosn, AZ 85712. Contact: Eric G. Ramsay, M.D. Approved for category 1 credit.

Family Practice Department Meeting
1st Monday, 12 Noon, Contact: R. Grossman, M.D.

GOOD SAMARITAN HOSPITAL

1033 East McDowell Rd., Phoenix, AZ Contact: Warren A. Hinchcliffe, M.D. Approved for category 1 credit.

Anatomical Path. & Cytology
Weekly, Mondays 4:00-5:00 P.M., S-4 Conf Rm.
Gynecological Section Conference
2nd Monday, 12:30-1:30 P.M., West Rm
Monday Noon Conference
Weekly, 12:00-1:00 P.M. R-2 Conf. Rm.
Mortality/Morbidity, Surgery Comm.
2nd Monday, 11:30-2:00 P.M., West Rm.
Obstetrical Section Conference
1st Monday, 12:30-1:30 P.M., West Rm.
Pulmonary Disease Grand Rounds
Weekly, 12:00-1:00 P.M., D-5 North
Family Practice Conference
Weekly Tuesday, 12:00-1:00 P.M., Fam. Practice Conf. Rm.

Pediatric Grand Rounds
3rd. Tuesday, 7:300-8:30 A.M., West Rm.

Surgical Grand Rounds
Weekly, Tuesday 7:00-8:30 A.M. East Rm.

Clinical Cancer Fourm
3rd Wednesday, 12:00-1:00 P.M., West Rm, Contact: John A. Bruner, M.D.

Combined Cardiovascular & Family Practice Conf.
Ist, 2nd, & 4th Wed., 12:00-1:00 P.M., D-5 North.

Dr. Finger's Lecture
Weekly, Wed., 12:30-1:30 P.M., R-2 Conf. Rm.

Learning Disabilities
Weekly, 10:30-12:30 A.M., R-2 Conf. Rm.

OBGYN Dept. Review & Statistics
Weekly, 12:30-3:00 P.M., OBGYN Conf. Rm.

PM & R Grand Rounds
1st. & 3rd. Wed., 12:00-1:00 P.M., R-2 Conf. Rm.

Gynecological Pathology
Weekly Thursday, 1:00-2:00 P.M., S-4 Conf. Rm.

GI Radiology Conference
1st Thursday, 12:00-1:00 P.M., F151, Radiology Conf. Rm.

Phoenix Ultrasound Soc. Meeting.
Medical Education Building, Contact: Dr. Ben Ora

Neurology Grand Rounds
Weekly Thursday, 9:00-10:00 A.M., D-5 North.

Tumor Treatment Planning Conf.
2nd & 4th Thursdays, 12:00-1:00 P.M., F151 Radiology

Joint Tumor Gyn Conference
2nd Friday, 12:30-1:30 P.M., West Rm.

Kinesology
Weekly Friday, 4:00-5:00 P.M., R-2 Conf Rm.

Medical Grand Rounds
Weekly Friday, 8:00-9:00 A.M., West Rm.

Neuropathy Conference
Last Friday, 1:15-2:15 P.M., S-4 Morgue.

Psychiatry Grand Rounds
Weekly Friday, 11:30-1:00 P.M. , East Rm.

PM & R Journal Club
Alt. Fridays, 12:00-1:00 P.M., R-2 Conf. Rm.

KINO COMMUNITY HOSPITAL

2800 E. Ajo Way, Tucson, AZ 85713. Contact: Eric G. Ramsay, M.D. Approved for category 1 credit.

Attending's Hour
Weekly, 12:30 P.M., Contact: Joseph Goldberger, M.D.

Infectious Disease Conference
3rd Monday, 12:30 P.M., Contact: Barry Friedman, M.D.

Nephrology Conference
1st Monday, 12:30 P.M., Contact: Alan Cohn, M.D.

OB/GYN Dept. Meeting
Weekly Mondays, 7:30 A.M., Contact: Lewis Shenker, M.D.

Psychiatric Conference
2nd & 4th Mondays, Contact: Jose Santiago, M.D.

Surgical Conference
Weekly Mondays, Contact: Ronald Fischer, M.D.

Cardiology Conference
4th Tuesday, 12:30 P.M., Contact: Brendan Phibbs, M.D.

Gynecology Conference
5th Tuesday, 12:30 P.M., Contact Lewis Shenker, M.D.

Neurology Conference
1st & 3rd. Tuesday, 12:30 P.M., Contact: H.W. Buschsbaum, M.D.

Radiology Conference
2nd Tuesday, 12:30 P.M., Contact: Marvin Baker, M.D.

Clinical Pharmacology
4th Wed., 12:30 P.M., Contact: Lee Gardner, Pharm. D.

Clinical Pathology Conf.
2nd Wed., 12:30 P.M., Contact: Jay Fleishman, M.D.

Gynecology Pathology Conference
Weekly Wed., 7:45 A.M., Contact: Lewis Shenker, M.D.

Medical Conference-Mortality & Morbidity
3rd Wed., 12:30 P.M., Contact: Joseph Goldberger, M.D.

Rheumatology Conference
1st Wed. 12:30 P.M., Contact: George Werner, M.D.

Cardiology Conference
1st Thursday, 12:30 P.M., Contact: Brendan Phibbs, M.D.

Clinical Correlation
3rd Thursday, 12:30 P.M., Contact: Elliot Munjack, M.D.

Endocrine Conference
2nd Thursday, 12:30 P.M., Contact Elliot Munjack, M.D.

OB/GYN Dept. Metting
2nd Thursday, 2:00 P.M., Contact: John Wurzel, M.D.

G-I Conference
4th Thursday, 12:30 P.M., Contact: Ray Moldow, M.D.

OB/GYN Sonography/X-ray Conference
4th Thursday, 3:00 P.M., Contact: John Wurzel, M.D.

Chest Conference
2nd & 4th Fridays, 12:30 P.M., Contact: David Engelsberg, M.D.

Dermatology Conference
3rd Friday, 12:30 P.M., Contact: Robert A. Schwartz, M.D.

Hematology Conference
1st Friday, 12:30 P.M., Contact: Steven Ketchel, M.D.

Medical Conference
5th Friday, 12:30 P.M., Contact: Joseph Goldberger, M.D.

MARYVALE SAMARITAN HOSPITAL

5102 W. Campbell Ave., Phoenix, AZ 85008

Continuing Medical Education Program
Every other Wednesday, 7-8 A.M.

MARICOPA COUNTY GENERAL HOSPITAL

2601 E. Roosevelt, Phoenix, Az 85008. Contact: Steven Schaffner, M.D.

Medicine Conference
Daily 12:00-1:00 P.M., Contact: Steven Schaffner, M.D.

Chest Surgery Conference
Weekly Mondays, 1:30 P.M., Santa Cruz Rm.

OB/GYN Departmental Grand Rounds
Weekly, 12 Noon, Santa Cruz Room.

Obstetrical Problem Conference
Weekly Tuesday, 7:15 A.M., Yavapai Rm

Orthopedic Conference
Weekly Tuesday, 7:30 A.M., Santa Cruz Rm.

Pediatric Grand Rounds
2nd Tuesday, 7:30-8:30 A.M.

Urology Discharge Planning Conference
Weekly,11:30 A.M., Station 42

Hand Surgery Conference
Weekly Wed., A.M., Santa Cruz Rm.

Neurosurgery Discharge Planning
Weekly Wed., 1:30 P.M., Station 42.

OB/Perinatology Seminar
Weekly, 7:15 A.M., Yavapai Room

OB/Neonatology Seminar
Weekly Wed. 7:15 A.M., Yavapai Room

Clinical Psychiatric Conference
Weekly, Wed., 11:00-12:00 P.M., Mental Health Annex, Rm 1346

Surgery Conference
Weekly Wed., 7:00-8:00 A.M., Surgical Dept.

Cardiology Conference
Weekly Thurs. 2:00 P.M., Santa Cruz Room.

OB/GYN Resident Conference
Weekly Thurs. 12:00 P.M., Yavapai Rm.

GYN, Endocrine Seminar
1st. Friday, 12:30 P.M., Santa Cruz Rm.

GYN Grand Rounds
Weekly Friday, 1:30 P.M., Santa Cruz Rm.

OB/GYN Surgical Pathology Conf.
Weekly Friday, 7:00 A.M., Yavapai Rm.

Orthopedic X-Ray Conference
Weekly Friday, 7:30 A.M., Santa Cruz Rm.

PHOENIX BAPTIST HOSPITAL & MED. CENTER

6025 N. 20th Ave., Phoenix, AZ 85015. Contact: James L. Grobe, M.D. Approved for category 1 credit.

Medical & Surgical Conference
Weekly Monday, 12:00 Noon

Medical Conference
1st. & 3rd Tuesdays, 12:00 Noon.

Medical & Surgical Conference
Weekly Wed., 12:00 Noon.

Medical & Surgical Conference
Weekly Friday, 12:00 Noon.

OB/GYN Section Conference
3rd & 4th Mondays, 1st Floor Conference Room

PHOENIX INDIAN MEDICAL CENTER

4212 North 16th St. Phoenix, AZ 85016. Contact: Leland L. Fairbanks, M.D. Approved for category I credit.

Pediatric Grand Rounds
1st Tuesday, 7:30-8:30 A.M., Pediatric Department

Clinical Staff Teaching Conference
Weekly Wed., 8:00-9:00 A.M., Conf. Rm. A.

PHOENIX MEMORIAL HOSPITAL

1201 S. 7th Ave. Phoenix, AZ. 85036. Contact: George Scharf, M.D., Approved for Category 1 credit.

Monthly Medical Education Seminar
3rd Monday, 6:30 P.M., Kiva Conf. Rm.
Tumor Board Conference
Weekly Friday, 12:00 P.M., Kiva Rm., Contact: Howard Kimball, M.D.

SCOTTSDALE MEMORIAL HOSPITAL

7300 East 4th St., Scottsdale, AZ 85251. Contact: W. S. Williams, M.D. Approved for category 1 credit.

Family Practice Conference
1st Monday, 12:30 P.M., Doctor's Lounge
Morbidity & Mortality Conference
2nd Monday, 12:30 P.M., Doctors' Lounge
Neurology & Neurosurgery Conference
3rd Monday, 12:30 P.M., Doctors' Lounge
Internal Medicine-Clinical Pathlogical Conference
4th Monday, 12:30 P.M., Doctors' Lounge
Pulmonary Medicine Conference
1st Tuesday, 12:30 P.M., Doctors' Lounge
Cardiology Conference
2nd Tuesday 12:30 P.M., Doctors' Lounge
Surgery Departmental Meeting
3rd Tuesday, 12:15 P.M., Doctors' Lounge
Thoracic & Vascular Surgery Conference
4th Tuesday, 12:30 P.M., Doctors' Lounge
Cardiopulmonary Conference
4th Tuesday, 12:30 P.M., Doctors' Lounge
OB/GYN Depart. Meeting & Conference
2nd Wed., 12:30 P.M., Doctors' Lounge
Trauma Conference
3rd Thurs., 12:30 P.M., Doctors' Lounge
Tumor Conference
4th Thursday, 12:30 P.M., Doctors' Lounge
Psychiatry & Behavioral Sciences Conference
1st Friday, 12:30 P.M., Doctors' Lounge
GI Medical-Surgical-Radiological Conference
2nd Friday, 12:30 P.M., Doctors' Lounge
Uronephrology-Oncology
Last Friday, 12:30 P.M., Doctors' Lounge

ST. JOSEPH'S HOSPITAL PHOENIX

350 West Thomas Road, Phoenix, AZ 85013. Contact: Joseph C. White, M.D.

OB/GYN Section Conference
3rd & 4th Mondays, 12:30-1:30 P.M., 1st Floor Conf. Rm.
Medical Grand Rounds
Weekly, Wed., 8:00 A.M., 1st Floor Conference Rm.
Pediatric Grand Rounds
4th Tuesday, 7:30-8:30 A.M., Contact: J. Kipp Charlton, M.D.

ST MARY'S HOSPITAL & HEALTH CENTER

1601 W. St. Mary's Road, Tucson, AZ 85703. Contact: see below.

Monthly Specialty Conference-Dept. of Surgery
1st Monday, 7:30 A.M., Century Rm. A. Contact: Med. Staff Office.
Grand Rounds: Medical Surgical, Family Practice, Pathology, Radiology
Weekly Thursday
Cardiology Conference
Weekly Friday, 8:00-9:00 A.M., Century Rm. Contact: Anthony Forte, M.D.

TUCSON MEDICAL CENTER (THMEP)

5301 E. Grant Road, Tucson, AZ 85716. Contact: Eric G. Ramsay, M.D. Approved for Category 1 credit.

Cardiology Conference
Weekly, 12 Noon, Contact: J. Fernadez, M.D.
Dermatology Conference
4th Monday, 5:15 P.M., Contact R. Friedman, M.D.
Perinatal Conference.
2nd Monday, 7:30 P.M., Contact: J. Lohman, M.D.
Hematology Conference
2nd, 3rd, 4th Tuesday, 12 Noon, Contact: M. Boxer, M.D.
Pediatric Grand Rounds
1st & 3rd Tuesday, 12 Noon, Contact: H. Thompson, M.D.
Cardiac Cath. Conference
2nd & 4th Wednesday, 5:15 P.M., Contact: L. Buckels, M.D.
Medical Conference
Weekly Wednesday, 8:00 A.M., Contact: M. Fuchs, M.D.
Neurology-Neurosurgery Conference
Weekly Wednesday, 12 Noon, Contact: H. W. Buchsbaum, M.D.
Chest Conference
3rd. Thursday, 12 Noon, Contact: Wm. Nevin, M.D.
Dermatology Conference
1st Thursday, 12 Noon, Contact: R. Friedman, M.D.
Infectious Disease Conference
2nd Thursday, 12 Noon, Contact: B. Friedman, M.D.
Nephrology Conference
4th Thursday, 12 Noon, Contact: S. Seltzer, M.D.
Neuroradiology Conference
Weekly Thursday, 5:00 P.M., Contact: N. Komar, M.D.
Surgical Literature Course
2nd Thursday, 7:00 P.M., Contct: P. Whitney, M.D.
Tumor Conference
Weekly Thursday, 12 Noon.
G-I Conference
Weekly Friday, 12 P.M., Contact: C. Krone, M.D.
Inter-Hospital Nuclear Medicine Conference
Weekly Friday, 7:15 A.M., Contact: S.V. Hilts, M.D.
OB/GYN Conference
1st Friday, 7:30 A.M., Contact: P. Evans, M.D.
OB/GYN Pathology Conference
3rd Friday, 7:30 A.M., Contact: R. Spark, M.D.
Dept. of Surgery Conference
Weekly Friday (except 1st), 8:00 A.M., Contact: R. Hasting, M.D.
Surgical Grand Rounds
1st Friday, Contact: P. Whitney, M.D.

U OF A HEALTH SCIENCES CENTER

Sponsor: U of A College of Medicine, Tucson, AZ 85724. Contact: See below. Approved for category 1 credit.

Anesthesiology Board Review Conference
2nd & 4th Monday, 4:00-5:00 P.M., AHSC Dining Rm. C&D, Contact: Dr. Vaughn & Kryc.
Anesthesiology Basic/Clinical Sciences Lectures
Weekly Thursday, 4:00-5:00 P.M., Room 5403
Anesthesiology Case Discussion
Weekly Wed., 7:00 A.M., AHSC, Dining Rm. C&D.
Anesthesiology Resident Presentation
1st Monday, 4:00-5:00 P.M., AHSC Dining Room, C&D, Contact Drs. Otto & Zehngut.
Cardiac Catheterization Conference
Weekly Friday, 4:00 P.M., Contact: Dr. Temkin.
Cardiology Conference
Weekly Monday, 12:00 P.M., AHSC, Contact: Dr. J. Fernandez
Cardiology Research Conference
Weekly Tuesday, 7:30 A.M., Contact: Dr. Roeske.
Tucson Cardiovascular Society
1st Thursday, 6:00 P.M., AHSC, Contact: Dr. Byrne-Quinn
Cerebrovascular Disease Conference
Weekly Monday, 5:00-6:00 P.M., AHSC, Rm. 5505, Contact: Dr. W. Moore
Clinical Immunology, Allergy & Rheumatology Rounds
Every Friday, Noon-1 P.M. Contact: John Boyer, M.D., Dept. of Internal Medicine.
Cerebrovascular Disease Conference
Mondays, 5-6 P.M., Weekly, Rm. 5505. Contact: Wesly Moore, M.D.,Depts. of Surgery, Nurology & Radiology.
Dermatology Conference
4th Monday, 5:15 P.M., AHSC, Contact: Dr. R. Friedman.
Dermatology Rounds
Weekly Wed., 11:30 A.M., Contact: Dr. Lynch
Endocrinology Seminar
Weekly Thursday, 12:00-1:00 P.M., Contact: Dr. Johnson
GI Pathology Conference
4th Friday, 1:30 P.M., AHSC, Contact: S. Paplanus
GI Radiology Conference
2nd. & 4th Mondays, 7:30 A.M., AHSC, Contact: Dr. T. Hunter
Head & Neck Tumor Management Conference
Weekly Wed., 4:00 P.M., Contact: Dr. Manning
Hematology-Oncology Clinical Conference
1st & 5th Tuesdays, Noon-1 P.M., Contact: M. Boxer, M.D., Room 6505, Dept. of Internal Medicine.
Medical Grand Rounds
Weekly Wed., 12:00-1:00 P.M., AHSC, Contact: Dr. J. Smith.
Morbidity/Mortality in E.M.
2nd Tuesday, 9:00 A.M., AHSC, Contact: Drs. Hughes & Alcorn
Neuromuscular Disease Conference
Weekly Friday, 11:30 A.M., Contact: Dr. Stern
Neuropathology Review Conference
Thursdays, 8:30 A.M., Rm. 5120, UAHSC.

Neuroradiology Conference
Weekly Thurs., 5:00 P.M., Contact: Dr. P.C. Christenson
Neuromuscular Journal Conference
2nd & 4th Thursday, 7:00-9:00 P.M., Contact: Dr. Stern.
Neuropathology Case Review
Weekly Friday, 8:30 A.M., AHSC, Contact: Dr. P. Johnson.
Neurosciences Seminar
Weekly Tuesday & Friday, 7:30 A.M., AHSC, Contact: Dr. C. Bamford
Nuclear Medicine
Weekly Thursday, 7:30 A.M., AHSC Radiology Conference Room
Ophthalmology Grand Rounds
Weekly Friday, 8:00 A.M., AHSC, Contact: Dr. H. Cross
Ophthalmology Retina & Fluoro. Conference
Weekly Thursday, 5:00 P.M., AHSC Contact: Dr. H. Cross
Orthopedic Conference
1st Tuesday, 7:30 P.M., TMC. Contact: Dr. L. Mann
Orthopedic Rounds
Saturday, 8:00 A.M., Contact: Dr. Peltier.
Pain Conference
3rd Monday, 4:00-5:00 P.M., AHSC Dining Rm. C&D, Contact: Drs. Hameroff & Cork.
Pathology Seminar
Weekly, 4:30-5:30 P.M., AHSC, Room 5120. Contact: Dr. P. Finley
Clinical Pathology Conference
5th Thursday, TMC, Contact: Dr. M. Fuchs
Tucson Pathologist Conference
1st Monday, 7:30 P.M., AHSC, Contact: Dr. A. R. Graham.
Pediatric Grand Rounds
2nd & 4th Tuesdays, 12:00 P.M., AHSC, Contact: Dr. H. Thompson.
Pediatric Problem Patient Conference
Weekly Wed., 8:00 A.M., Contact Dr. Walson
Pediatric Research Forum
Weekly Tuesday, 7:30 A.M., Contact: Dr. James Jones
Pediatric Specialty Conference
Weekly Friday, 8:00 A.M., Contact: Dr. Philip Walson.
Peri-Natal Conference
2nd Monday, 7:30 P.M., AHSC, Contact: Dr. J. Lohman.
Psychiatric Grand Rounds
Weekly Wed., 4:00 P.M., Contact: Dr. R. Cutts
Psychiatric Monthly Case Conference
2nd Friday, 7:30 A.M., Contact: Dr. Alan Levenson,Palo Verde Hospital
Pulmonary Rounds
Weekly Friday, 11:30 A.M., Contact Dr: Benjamin Burrowns.
Chest Radiology
Weekly Monday, 5:00-6:00 P.M., Rm 1535F, UAHSC. Contact: Irwin M. Freundlich, M.D., Dept. of Radiology.
Neuroradiology Teaching Conference
Weekly Wednesday, 7:30 A.M., AHSC, Contact: Dr. Christenson
Radiation Oncology Planning Conference
Weekly Friday, 8:30-10:00 A.M., AHSC, Room 0655
Radiology Interesting Case Conference
Weekly Thursday, 12:00 Noon, AHSC, Contact: Dr. Freundlich
Gastrointestinal Radiology
Weekly Tuesday, 5:00-6:00 P.M., AHSC, Contact: Radiology Dept.
Radiology-Rheumatology Conference
Weekly Thursday, 7:45 A.M., UAHSC, Library Rm.1535C. **Uroradiology (Genitourinary Radiology)**
2nd & 4th Mondays, 7:30- 8:30 A.M., AHSC, Contact: Dept. of Radiology
Renal Pathology Conference
1st & 3rd Thurs., 11:30 A.M., Contact: Dr. Nagle.
Residents Noon Conference
Weekly Tuesday & Thursday, 12:00 Noon, AHSC, Contact: Dr. A. Greensher.
Resident's Conference
Wednesday, weekly 5-6 p.m., Diag. Radiology Conf. Rm.
Surgical Morbidity & Mortality Conference
Weekly Wed., 8:00 A.M., Contact: Dr. Wangensteen.
Joint Thyroid Conference
1st & 3rd Wed., Contact: S. C. Werner, M.D.
Trauma Conference
Every Thursday,4-5 p.m. UAHSC, Rm. 5505.
Ward Grand Rounds - Trauma
Weekly Tuesday, 8:00 A.M., Contact: Dr. M. Silverstein.
Toxicology Conference
Weekly Tuesday, 8:00 A.M., Contact: Dr. Keith Likes
Section.
Tucson Ultrasonography Group
Weekly Wed., 4:30 P.M., AHSC, Contact: Dr. I. Freundlich.
General Urology Conference
Weekly Tuesday, 12:00 Noon, AHSC & VA Hospital Contact: Dr. G. W. Drach.

VETERANS ADMINISTRATION MEDICAL CENTER

7th St. and Indian School Rd., Phoenix, AZ 85012. Sponsor: VA Hospital. Contact: Alfred Heilbrumn, M.D. Approved for Category 1 credit.

Cancer Symposium
Weekly Wed., 1:00 P.M., Contact Dr. Byrne.
Cardiology Conference
2nd Thursday — 1 p.m. — T5, Dr. Habibzadeh, Ext. 348.

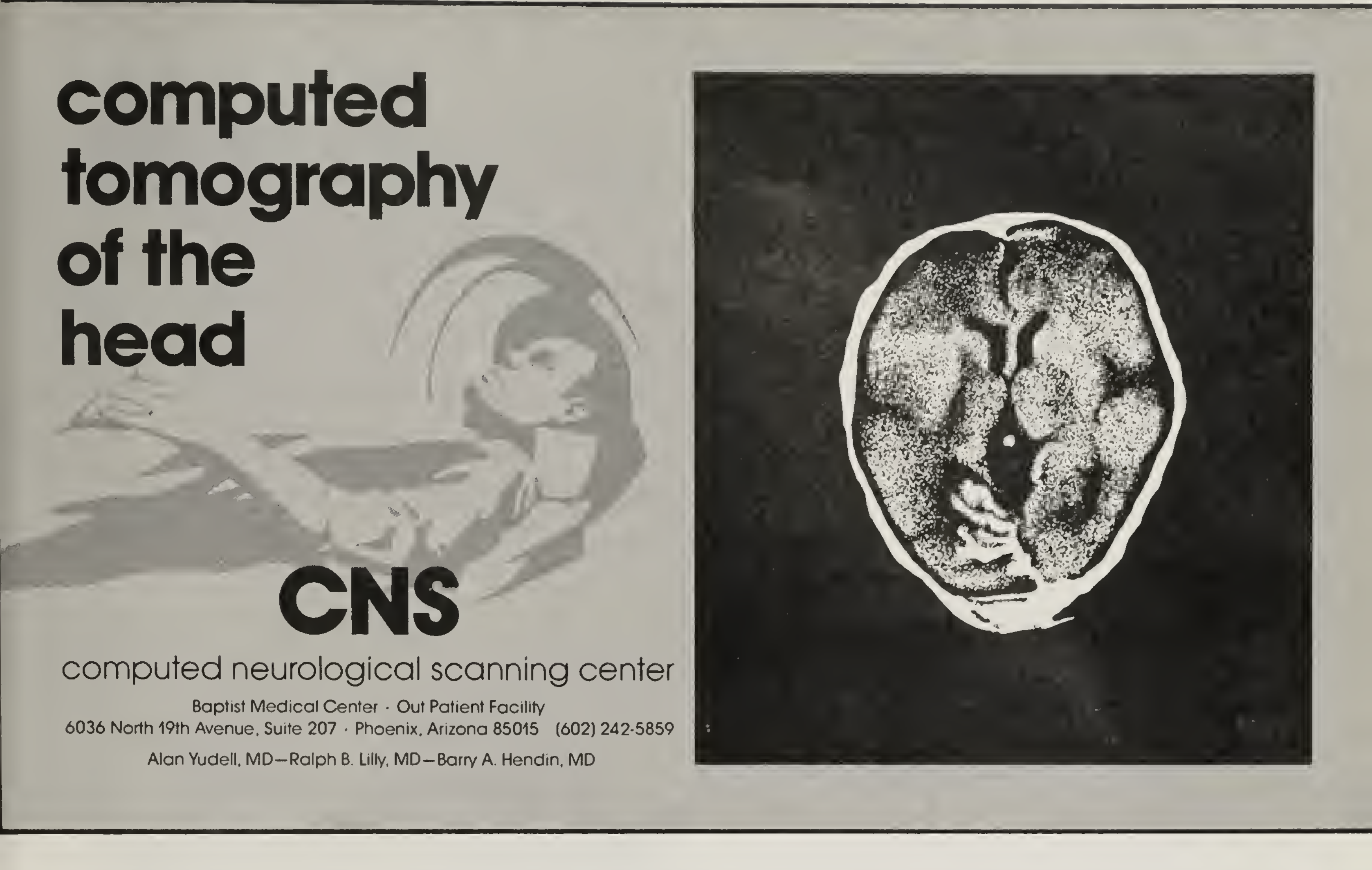
Dermatology Conference
1st, 2nd, & 3rd Wednesday 10 A.M. — C118, Dr. Roberts, Ext. 426.

G.I. Clinic
2nd & 4th Wed, 1:00-4:00 P.M., GI Ward
G.I. Radiology Clinical Correlation Conference
1st & 3rd Monday,1:00 P.M., D435.
G.I. Grand Rounds
Weekly, Wed., 8:00 A.M., G-I Ward , Contact: Dr. Severino
G.I. Resident Conference
Weekly Friday, 1:00 P.M., D446, Contacts: Drs. Sanowski & Kozarek
G.I. Rounds
Weekly Thursday, 1:00 P.M., D446
Medical Service Grand Rounds
Weekly Friday — 11 a.m. — T5, Dr. Zeller, Ext 272
Medical-Surgical G.I. Conference
1st & 3rd Monday, 3:00 P.M., Room 3134
Medical-Surgical G.I. Conference
1st & 3rd Friday, 2:00 P.M., Rm. 2128, Contact: Drs. Kozarek and Mertz, Ext 493
Medical-Surgical Chest Conference
1st and 3rd Thursday — 1 p.m. — T5, Dr. Rohwedder & Dr. Gill.
Surgical X-Ray Conference & Bedside Teaching Rounds
Weekly, Monday, Room 3134.
Surgery-Pathology Conference
Weekly Thursday, 2 p.m. — 2128, Drs. Mertz, Ext. 493 & Likos, Ext 623

TUCSON VETERANS ADMINISTRATION MEDICAL CENTER

3601 South Sixty Ave., Tucson, AZ 85723. Contacts listed below. Approved for category 1 credit.

Medical Surgical Chest Conference
Weekly Tuesday, 2:00 P.M., Contact: Dr. Young
Medical Grand Rounds
Weekly Wed., 12:00-1:00 P.M., V.A. Hospital Staff Conf. Rm. & (AHSC), Contact: Jay Smith, M.D.
Surgical Grand Rounds
Weekly Wed., 4:00 P.M., Contact: Dr. Putnam
Endocrinology Seminar
1st.,3rd, & 5th Thurdsays, 12:00-1:00 P.M., Rm. N318, Contact: Dept. of Internal Medicine.
Vascular Radiology, Interesting Case Conf.
Weekly Thursday, 12:00 Noon
Neurology Grand Rounds
Weekly, Friday, 12:00 P.M., Contct: Dr. Sibley.



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Today there are over 31 million men, women and children suffering from the many forms of arthritis and related rheumatic diseases. And there just aren't enough trained rheumatologists to help them.

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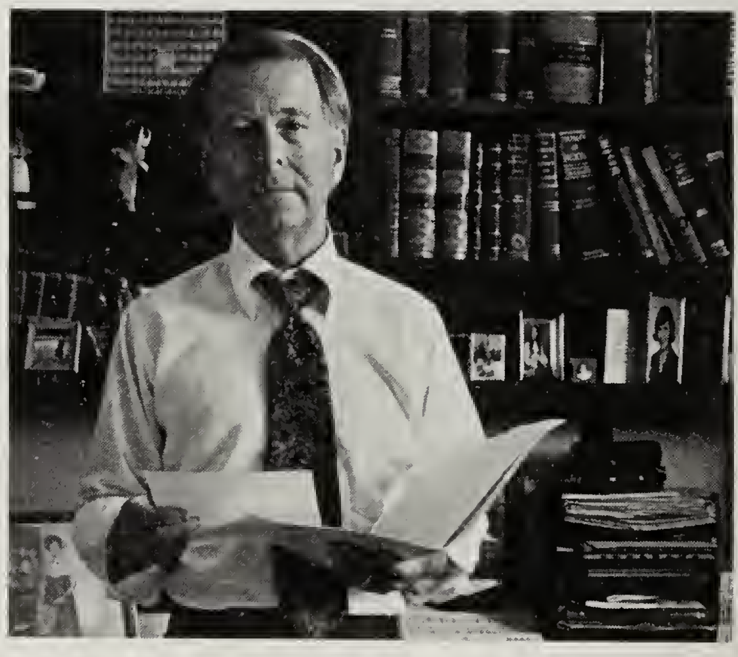

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Physician wanted to direct major section in Dept. of Family & Community Medicine. Professional rank, position requires national, international activities, health care administration and managment, coummunity development, graduate/undergraduate education. Responsibilities include directing general preventive medicine, residency program. Send resume to: Search Committee A

UNIVERSITY OF ARIZONA, FACULTY POSITIONS: ALL LEVELS, FAMILY & COMMUNITY MEDICINE.

Southwestern university hospital in large health sciences center now recruiting primary care physicians, specialists in family/community medicine. Unique opportunities in research, graduate/undergraduate education and patient care. Send resume to: Search Committee B.

SUN BELT, UNIVERSITY OF ARIZONA, DIRECTOR: FAMILY PRACTICE SECTION

Southwestern university seeking Director for Family Practice Section at university hospital in health sciences center.

Responsibilities include directing model practice unit, approved Family Practice Residency and undergraduate programs at the clerkship and preceptorship levels. Must be clinically active and board certified in Family Practice. Send resume to Search Committee C.

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All inquiries are confidential. Send all resumes to:

Search Committee (A, B, or C)
Dept. of Family & Community Medicine
University of Arizona Health Sciences Center
Tucson, AZ 85724

FINANCIAL

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OB/GYN

Completing OB/GYN residency 6/80. Interested in joining active practice. Will consider group or solo. Frederick D. Sanders, M.D., 1908 Revere Road, Cleveland Heights, Ohio 44118. Phone (216) 371-4517.

PEDIATRICS POSITION

Needed full-time physician—completed specialty in Pediatrics. Position located at Santa Cruz County Health Department, Nogales, Arizona. Send resume to Dr. H.F. Pfister, Director, 200 La Castellana Drive, Nogales, Arizona 85621.

SEDONA

Needs FP's or Internist—Population 8,000 in Arizona's most beautiful and growing town, now underserved by only three FP's, situation partly relieved by specialists in Cottonwood, and Flagstaff, but excellent opportunity for well-rounded primary practitioneers ready for life in sophistcated small town, within easy reach of almost all other Arizona attractions. Excellent hospital 20 miles away in Cottonwood. I am moving into a new office complex and will make my present downtown 1200 square foot office, with or without 450 foot annex, available on open-ended terms, with move to built-to-suit quarters in new office possible later, with or without some type of association. If interested, plese contact Richard Griesenbeck, M.D., P.O. Box N, Sedona, AZ 86336. Phones (602) 282-7166 or 282-7349.

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